FEBRUARY, 1951

The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Some Unusual Forms of Perforation Complicating Benign Peptic Ulcer

Acute Perforation of Gastric and Duodenal Ulcers

Parenteral Fluids in the Surgical Patient

Primary Torsion of the Omentum

●

Sixteenth Annual Convention
Chicago, Ill., 17, 18, 19 September 1951

VOLUME 18 NUMBER 2

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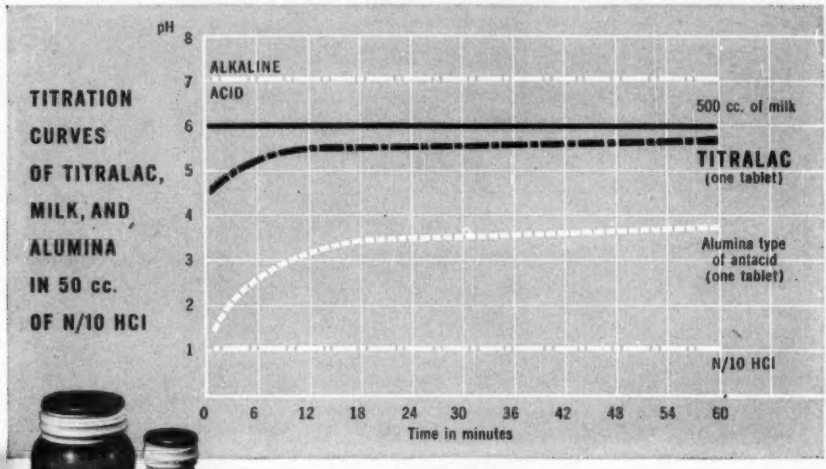

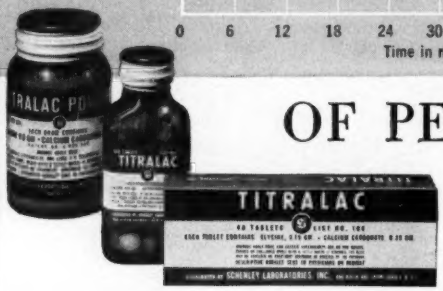

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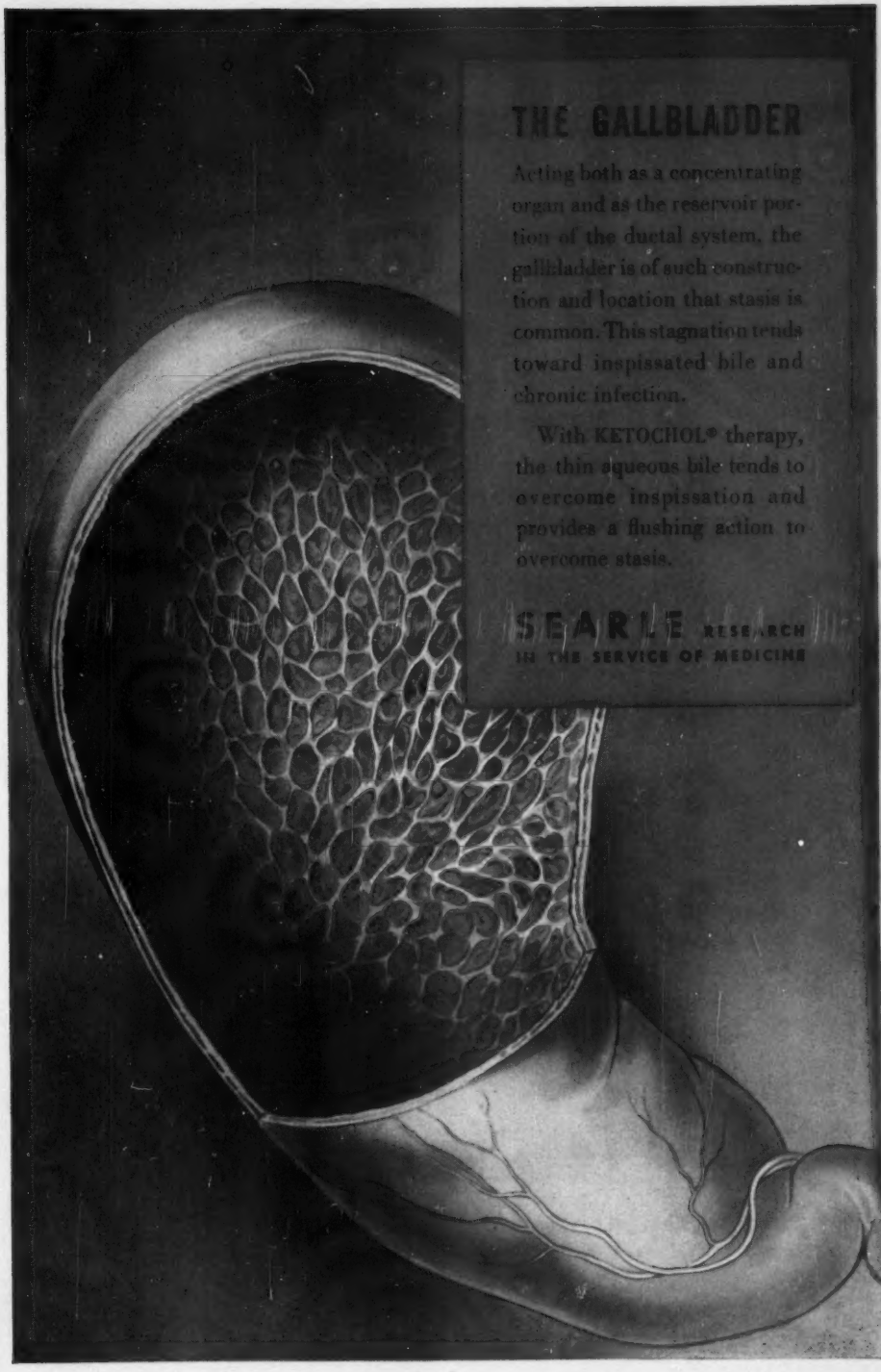
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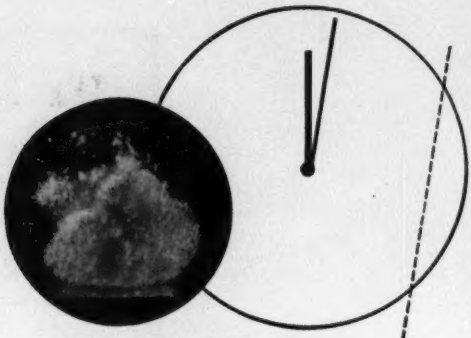

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(INCORPORATING THE AMERICAN JOURNAL OF GASTROENTEROLOGY)

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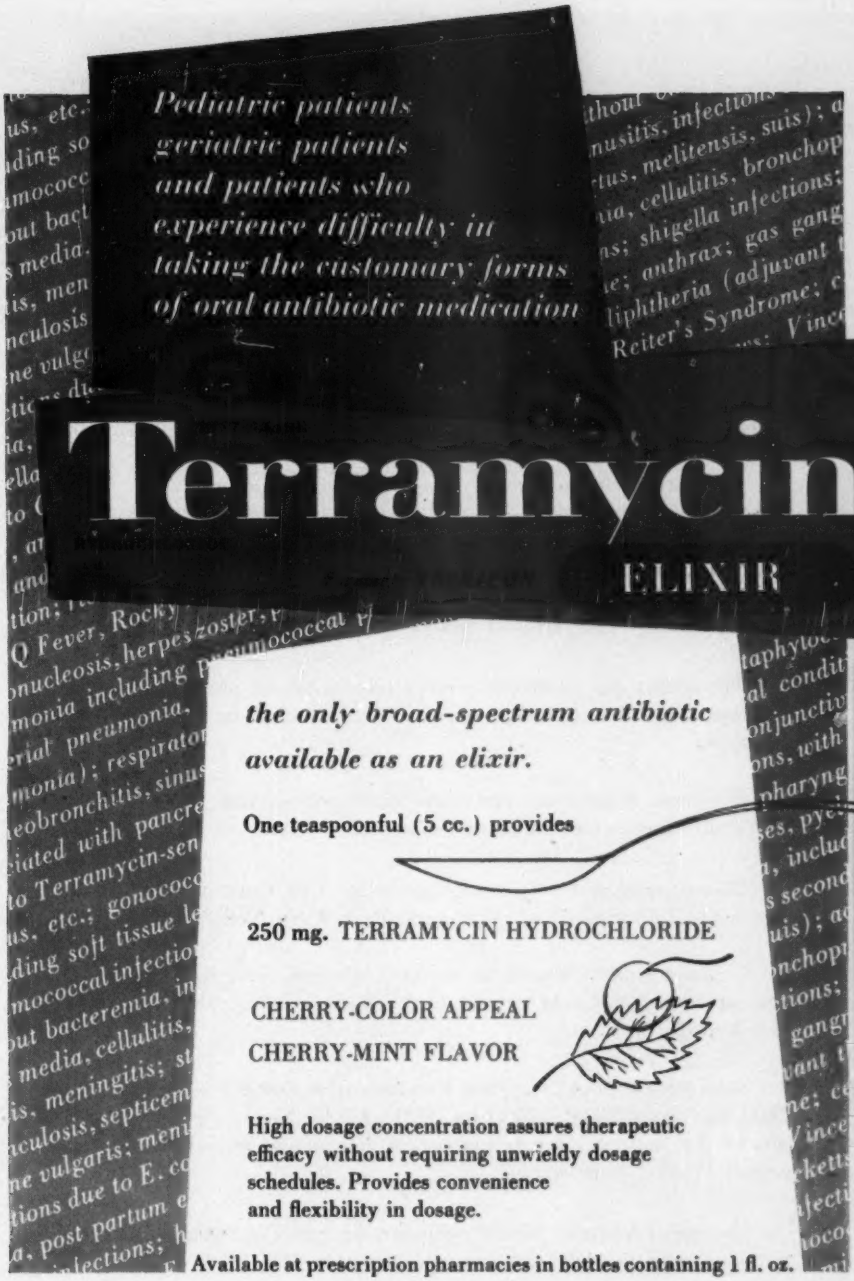

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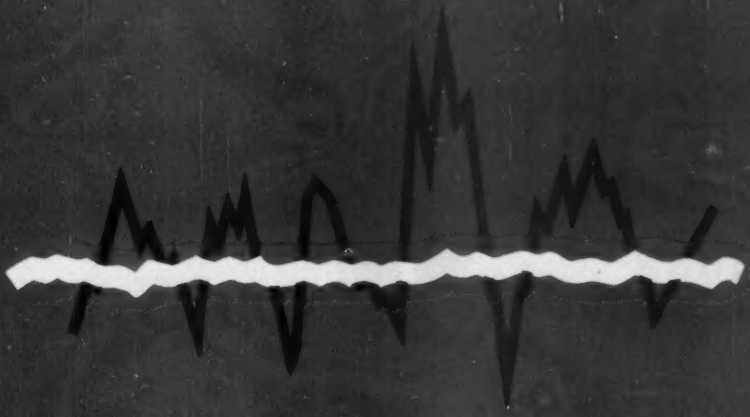

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The Review of Gastroenterology

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A monthly journal of Gastroenterology, Proctology and Allied Subjects

Volume 18 FEBRUARY, 1951 Number 2

A REVIEW OF SOME UNUSUAL FORMS OF PERFORATION COMPLICATING BENIGN PEPTIC ULCER*

MAURICE FELDMAN, M.D.

Baltimore, Md.

Unusual forms of perforation complicating peptic ulcer are not too often recognized. A review of the literature reveals but little information regarding perforations other than the diffuse progressive type involving the greater peritoneal cavity. Five types of perforations may occur as a complication of peptic ulcer: (1) those involving the greater peritoneal cavity, (a) diffuse, (b) formes fruste, (2) localized walled-off pocketing, (a) pseudodiverticular, (b) separated pocket adjacent to the perforation, (c) pocket distant from the perforation, (3) lesser peritoneal cavity, (4) retroperitoneal, and (5) into other organs. The acute variety of perforation with involvement of the greater peritoneal cavity and the chronic fistulous types will not be considered in this presentation.

The "formes fruste" perforation produce an acute abdomen, with initial symptoms similar to those of the acute type. There is board-like rigidity of the abdomen and the prostration accompanied with pain, but it is not as severe as in the classical case. The symptoms in this condition often subside in from 2 to 10 hours, leaving the patient in comparative comfort. The diagnosis of this condition cannot always be confirmed. However, the roentgen demonstration of air beneath the diaphragms will often confirm the clinical diagnosis of a perforating viscus.

The localized walled-off perforation is not an uncommon complication of peptic ulcer. This form of perforation may be classified as follows: (1) accessory pocket of pseudodiverticulum, the ulcer crater forming part of the pocket, (2) chronic perforation into a walled-off pocket, (a) adjacent to the ulcer crater, (b) distant from the ulcer crater, (3) pinpoint perforation with only air escaping into the pocket, (a) sealed medical type, and (b) nonsealed surgical type.

The accessory pocket or pseudodiverticulum type is one of the more common varieties of nonsealed walled-off perforation. When complicating a gastric ulcer with the crater situated on the lesser curvature of the antrum, the walled-off pocket usually adjoins the ulcer crater, appearing as an extra large sized ulcer or diverticulum. Those situated in the duodeum usually arise on the inferior and greater curvature side of the base of the bulb. This form of perforation is fre-

*Read before the Fifteenth Annual Convention of the National Gastroenterological Association, New York, N. Y., 9, 10, 11 October 1950.

quently asymptomatic. However, they do not tend to heal and are thus prone to repeated secondary inflammatory changes often producing intractable symptoms refractory to medical treatment. These chronic perforated ulcers possess a large opening which readily fill with barium and air. The communication between the viscus and pocket is clearly demonstrated roentgenologically. Occasionally there may be some retention of barium in the gastric pocket, but a residue is infrequently observed in the duodenal pseudodiverticulum type.

An infrequent form of chronic duodenal ulcer perforation may present itself as a localized encapsulated subhepatic or periduodenal pocket. The pocket in this variety is separated from the ulcer and does not form part of the ulcer crater, thus differing from the pseudodiverticulum type. The site of the pocket in chronic walled-off perforation varies according to the location of the ulcer. When it occurs on the lesser curvature of the body of the stomach it usually lies adjacent to the ulcer crater. It is less apt to occur elsewhere because of the close relationship of the ulcerated area with the body of the pancreas. When the duodenum is involved the pocket may be situated as an offshoot from the ulcer forming a periduodenal sacculation, or it may be distantly located in the subhepatic area. Perforations occurring in the distal portion of the pylorus may be localized or the pocketing may be observed in the subhepatic area. The walled-off pocket is usually large sized and often irregular in contour. Since it readily fills with barium the pocket is more or less easily demonstrated in the roentgen examination. It may produce pressure on adjacent structures. This type of walled-off perforation does not tend to disappear and continuously refill with the contents of the viscus. Often, these cases are asymptomatic at the time of the examination, but sooner or later become a surgical problem.

Little consideration has been given to a type of perforation caused by a pinpoint leakage, presenting a small walled-off pocket filled only with gas. In this condition two types are noted, a medical and a surgical. In the medical type of walled-off sealed pinpoint perforation complicating peptic ulcer, there is an escape of a small amount of air, forming an air-bubble. No barium enters into the pocket, which may be situated close to the ulcer crater or distantly, in the subhepatic region. These patients are ambulatory and are not acutely ill. The condition is a medical problem, usually subsiding after treatment. In the surgical type of walled-off gas-pocket due to pinpoint perforation, the pocketing is more extensive than in those of the medical type. It is usually accompanied with an associated localized active peritonitis. The perforation may be sealed forming an abscess, or nonsealed, however most of them are nonsealed. The air-bubble is not readily absorbed in this type, since there is a continuous leakage of gas into the pocket. The symptoms are progressive and tend to increase in intensity, necessitating surgical intervention.

Clinically, the pinpoint perforation is characterized by an acute, subacute or insidious attack of abdominal distress varying from a mild form to one of marked severity. In the medical type, the usual ulcer symptoms, in most instances, are exaggerated. These patients do not obtain relief with food, soda, or antispas-

modics as quickly as they had before. At times food seems to increase the pain and discomfort instead of giving relief. There is persistent epigastric pain which lasts longer than usual. Vomiting frequently occurs during the episode. The small subhepatic air-bubble tends to disappear with relief of symptoms after appropriate medical therapy. It must be emphasized here, that clinical symptoms may be absent in all forms of sealed or nonsealed walled-off perforations.

The sealed medical type of perforation must occur with greater frequency than has been suspected, but it has not been hitherto recognized. The ulcer is usually active, most often presenting roentgenologically a niche defect. The small gas-bubble can be more clearly demonstrated in the erect position during spot film compression examination; while in the recumbent prone position no air-pocket may be observed. In many cases the gas-bubble cannot be seen under the fluoroscope in any position, because of the minimal amount of air in the pocket. In the sealed type with a small air-bubble, the air is usually absorbed. It is therefore likely that many cases of the mild sealed pinpoint medical type of perforation occur and are missed because the examination is made at a time after the air had been absorbed. In the absence of the characteristic gas-pocket the diagnosis of this form of perforation cannot be made.

Lesser peritoneal cavity perforation has not been given much consideration and is not even mentioned in most textbooks. In a recent survey of this subject, a statistical study of 57 cases of lesser peritoneal cavity perforation was made. It was found to occur in 1 per cent of surgical cases of perforation of peptic ulcer. However, in our study of 1,522 autopsies not a single case of lesser sac perforation was encountered complicating benign peptic ulcer, though 2 cases were found to be due to perforation of a gastric carcinoma. In a recent communication a roentgen technic was proposed to demonstrate effusion and gas in the lesser peritoneal sac. Frequently, following perforation, air and fluid is observed in both the lesser and greater peritoneal cavities. This is due to a patent foramen of Winslow. With the usual roentgen technic the lesser sac involvement is not ordinarily demonstrated. When the foramen of Winslow is occluded the diagnosis is not often made because of inadequate roentgenographic studies. The roentgen technic for the demonstration of lesser peritoneal sac perforation is as follows: after taking the usual roentgenograms in the supine and anteroposterior erect positions, roentgenograms are then made in the lateral erect and in the right and left lateral supine and prone decubitus positions. If necessary a roentgenogram is also made in the Trendelenburg position. In the supine anteroposterior position, the lesser sac may be seen at times as a large rounded dense shadow conforming anatomically with the position of the lesser sac, lying between the stomach, liver, and left diaphragm. In the lateral erect view the sac is shown posteriorly, which best illustrates the air and fluid level. In the lateral supine and prone decubitus positions the fluid and air is shown to shift its position. The patency of the foramen of Winslow is of especial importance in the demonstration of effusion and gas in the lesser omental sac. Following a perforation, the lesser peritoneal sac fills and then overflows through the foramen, if patent, or may form an abscess within the sac.

The escape of air through the foramen of Winslow accounts for the frequency of observance of air in the greater peritoneal cavity. The foramen, however, may be occluded by inflammatory changes, and consequently the fluid and gas remains in the lesser sac. The roentgen signs may be variable, depending upon the amount of air and fluid in the sac, the amount of pressure made upon the adjacent organs and the patency of the foramen of Winslow. One or more roentgen signs may be demonstrated in the suspected case. The following roentgen signs indicate the presence of lesser peritoneal cavity involvement following peptic ulcer perforation: (1) visible dense mass, produced by accumulation of fluid in the lesser sac, (2) fluid level, capped by gas, (3) air beneath the diaphragms, (4) elevation of left diaphragm, (5) pressure defect on the posterior wall and lesser curvature of the stomach, (6) displacement of the stomach, (7) gastric walls pliable and flexible. (8) obliteration of stomach gas-bubble, (9) displacement of duodenum, (10) displacement of splenic flexure, (11) displacement of transverse colon, (12) preservation of the left kidney contour. In addition to the roentgen signs mentioned, secondary involvement of the thoracic cavity may occasionally be observed.

Retroperitoneal perforation complicating peptic ulceration is comparatively rare. When an ulcer is situated on the posterior wall of the distal two-thirds of the duodenum, it may perforate into the retroperitoneal cavity. This portion of the duodenum is located retroperitoneally; the anterior wall of the distal duodenum faces the greater peritoneum; the posterior wall faces the retroperitoneum. Clinically, the diagnosis is rarely made because of the vague symptoms encountered in this condition. The patient may exhibit few symptoms at the onset. There may be vomiting with some abdominal pain and backache. Later, following extravasation into the retroperitoneal tissues the symptoms become severe. A flat roentgenogram of the abdomen made in the supine position, will reveal evidence of air in the retroperitoneal spaces. A fairly accurate diagnosis of this complication is based upon the roentgen evidence of retroperitoneal emphysema. The emphysematous involvement follows the course of the retroperitoneal tissues and cavity. The duodenal contents and gas is dispersed along the root of the transverse mesocolon or mesentery or both, into the retroperitoneal space and over the kidneys, especially the right kidney. It follows the psoas muscle and retrocecal area from below, extending upward into the liver area, ribs and mediastinum. It may be complicated by a mediastinitis or pleurisy.

Summary

In this communication a review is made of the various forms of unusual perforations complicating peptic ulceration. The following types of perforations are presented: (1) formes fruste, (2) accessory pockets, (3) localized gas-pockets, (4) lesser peritoneal sac perforations, and (5) retroperitoneal perforation. Emphasis is made on the importance of recognizing the small air-bubble pinpoint perforations and the roentgen method for demonstrating them. Lesser peritoneal sac perforation and the roentgen diagnosis is discussed. Data is presented on retroperitoneal involvement following perforation of the posterior wall ulceration in

the distal duodenum. In the unusual forms of perforation, one must look for them carefully and consider the possibility of their occurrence, otherwise they may be overlooked.

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DISCUSSION

Dr. Frank J. Borrelli (Tuckahoe, N. Y.):—I think we owe a debt of gratitude to our distinguished speaker as I am sure he has made a contribution not only to our society but to medicine in general.

Most of you who are doing your work in hospitals realize the importance of many of these signs that Dr. Feldman has brought out. So often the patient is brought to the hospital with or without major symptoms of rupture, and reliance on the roentgenogram has, in my estimation, heretofore been over-emphasized; however, an example of how important this work that Dr. Feldman has presented is to us was experienced by me when a patient from South America was recently admitted to the hospital.

The films that were taken after the administration of barium showed, as Dr. Feldman demonstrated, a large, diverticulum-like, walled-off, sealed cavity just below the duodenal bulb. I noticed a very similar picture on one of the slides that Dr. Feldman showed. The "unfortunate" thing in this particular case was that the man was rather intelligent and told us that the operation in Venezuela was "connecting the gallbladder to his duodenum." The residents in the hospital immediately said this was a demonstration of a cholecystoduodenostomy, but to me it appeared rather high, and, being familiar with Dr. Feldman's work, I immediately thought of an old rupture in which the barium now filled into the sealed-off cavity.

He was operated upon and, of course, the cholecystoduodenostomy was not found.

In my estimation Dr. Feldman has presented us with many roentgenographic signs that we as radiologists and you as members of the services in gastroenterology, should be quite familiar with. The mere fact that gas can escape and be demonstrated beneath the diaphragm is very helpful when it occurs, I think, as Dr. Feldman has brought out, that you don't always find the gas beneath the right leaf of the diaphragm. It may only elevate the diaphragm and with your clinical picture you may have to go in on those two signs alone.

Most gastroenterologists, many of them good fluoroscopists, expect to see gas or gas fluid levels in one-projection, which I often find difficult, so that in our department we have a definite ruling that no single view or fluoroscopy alone is to eliminate perforations or suspicious perforations.

All patients, as Dr. Feldman has brought out, must be examined not only in the supine position in an anteroposterior projection, but must also have lateral views in the supine position as this is only a minimal study and one cannot be satisfied even with this.

As Dr. Feldman has told you, in the lateral supine position you may see the fluid level, and in the erect position you may see the stomach displaced forward; we therefore have a contribution in diagnosis that can be used by every gastroenterologist, who might otherwise take the attitude that these patients are terribly ill and probably be in the hospital in a moribund condition.

Many of you are familiar with the case that Dr. Bockus has reported in his book. The patient, having little or no symptoms following an injury, was ready to be discharged, when his temperature suddenly elevated, and they decided to go into the abdomen and found a three-inch rent in the stomach, with relatively no previous symptoms.

On the other hand with very tiny perforations in which the barium or food does not go through the perforation the presence of gas will give us a definite sign that we can use roentgenographically.

In conclusion in behalf of the various hospital services, especially the Radiological Department at our College, we are very grateful to Dr. Feldman for an excellent contribution in the more complete study of possible ruptures.

Dr. Hyman I. Goldstein (Camden, N. J.):—The helpful studies made by Dr. Feldman, of course, would be impossible without the great epoch-making contribution and discovery by Roentgen in November 1895. Further, many well-known persons would not have died from undiagnosed, misdiagnosed, or unsuspected perforations of gastric and duodenal ulcerations—were such studies available, and applied!—Rudolph Valentino; Jean D'Arcet, French chemist; Camillus Facinus—M. Donatus' patient; the Duchess of Orleans; the noted Cardinal Caesar Baronius; Jacopo Penada's patient; Horatius Augenius' patient; Rayer's case; Christopher Rawlinson's case of perforated gastric ulcer; J. C. Volcamerus' case; Morgagni's case of Tyson; Muralt's case, and Bonetus' report of John Bauhin's young female patient (18 years old), who died of perforated gastric ulcer—might have been avoided, with the present-day advances and these fine observations and studies made by Dr. Maurice Feldman!

Dr. Maurice Feldman (Baltimore, Md.):—During the war period my attention was first directed to acute episodes that occurred in many of our cases of duodenal ulcer. We were struck by the fact that our patients for many years have had these ulcers and were able to get relief with soda and food, and with a sudden episode, they found that their symptoms were reversed. They had vomiting, the pains were more severe, thus giving us the impression that there was some complication

present. Only by spot-filming these patients in the upright position, were we able to find the small pockets of gas, which enabled us to make the diagnosis and avoid an operation.

The purpose of my presentation was therefore to reemphasize the importance of recognizing these uncommon perforations which have hitherto not been recognized.

PARENTERAL FLUIDS IN THE SURGICAL PATIENT*

With Special Reference to Pre- and Postoperative Care in Serious Gastrointestinal Lesions

HENRY M. YOUNG, M.D.
New Britain, Conn.
and
JOHN M. McGOWAN, B.S., M.D., C.M., M.S., F.A.C.S., F.R.C.S.(C)
Boston, Mass.

The use of the parenteral route for the administration of fluids has become so universal that frequently insufficient attention is given to accurate determination of quantity and type of fluid indicated in each specific case. The material presented here is based on experience in handling seriously ill patients, both traumatic and postoperative in a large city hospital of 2,300 bed capacity. We have attempted to establish certain principles with which adequate and proper fluid and nutritional balance may be maintained in the seriously ill surgical patient by the parenteral route.

Correct fluid therapy requires: (1) adequate and complete diagnosis, (2) understanding of the pathological chemistry and physiology of the disease process, and basic requirements of the patient, and (3) realization of the potentialities of fluid therapy.

In this paper we shall discuss fluid therapy in relation to: (1) traumatic shock, (2) pre- and postoperative care of patients with serious gastrointestinal lesions requiring major surgery, and (3) cases presenting special surgical problems and complications.

Shock

Shock may best be defined as a form of peripheral circulatory failure in which there is a discrepancy between the volume of circulating fluid and the volume capacity of the circulating system. The causes of this discrepancy may or may not be associated with loss of fluid content.

Conditions in which there is no fluid loss such as occurs in association with vasodilatation may result from: (1) spinal anesthesia, (2) histamine, (3) alcohol and (4) acute or chronic cardiac failure.

Marked fluid loss occurs in (1) hemorrhage, (2) burns, (3) peritonitis, (4) intestinal obstruction and (5) crush injuries.

The *emergency treatment* of patients suffering from acute fluid loss consists of a rapid infusion preferably of glucose solution. This will sustain the patient until the blood can be typed and crossmatched for transfusion. Normal physiological saline has previously been the fluid most commonly employed, but because of a possible salt solution intolerance[1], and the dangers of pulmonary edema, we have found 5 per cent glucose solutions much more satisfactory. The proper *definitive* treatment of shock is replacement of the fluid lost in kind and amount. In the case of hemorrhage it is most important to determine the approximate amount

*From the Fourth Surgical Service, Boston City Hospital, Boston, Mass.

lost particularly if emergency surgery is contemplated. A 500 c.c. blood loss may be well tolerated and compensated for by the patient. Even with a loss of 1,000 c.c. the only warning signs may be a coolness of the extremities.

In treatment of shock due to blood loss, a rapid infusion of glucose or saline solution will elevate the blood pressure and maintain it for an hour or two; the effect, however, is not sustained. The fluid rapidly passes into the tissues. The effect of plasma is more lasting, and is, therefore, more suitable for the purposes of increasing the volume of the circulating fluid. However, because of the danger of homologous serum jaundice following plasma its use should be reserved for cases of extreme emergency where whole blood is not available. In acute hemorrhage one cannot rely on the hematocrit, hemoglobin and plasma protein determinations for an accurate estimate of blood loss since the lowering of these values depends on the process of hemodilution which may require 24 hours to become complete.

The proper and adequate treatment of *shock in cases of severe trauma* requires immediate mobilization of a coordinated group or *shock team*. It is only by this means that early shock can be detected in its incipient phase. While it is true that *one person* with available intravenous fluids can prevent an immediate fatality, *teamwork* can salvage a much higher percentage of cases particularly in dealing with abdominal injuries where prompt surgery is required. The high incidence of lacerated livers, ruptured spleens and ruptured viscera associated with fractures of the extremities has made us realize that without teamwork, multiple injuries cannot receive adequate therapy. The shock team should include: (1) the services of a technician or specially trained intern for typing and crossmatching of blood and other initial and repeated laboratory tests; (2) one intern or resident to take charge of administration of fluids, insertion of "cut downs" and preliminary examination and splinting of fractures; (3) one resident or visiting surgeon to make a complete and careful examination of the patient, and (4) a senior resident or visiting surgeon to supervise, receive reports, view x-rays and outline a plan of therapy.

Burns

In the case of *burns*, the blood pressure tends to be maintained at a higher level and for a longer period than in the case of shock due to trauma. Sweating is usually quite prominent and cyanosis may be present due to hemoconcentration. Emergency fluid therapy consists of giving 50 c.c. of plasma for each 1 per cent of the surface area of the body which has been burned[2]. Accepted figures for the various areas are: Head, 6 per cent; trunk, 38 per cent; upper extremities, 18 per cent; and lower extremities, 38 per cent. The hematocrit is above normal. In view of the marked drop in red count which usually occurs 48-72 hours after burn, additional blood should be given after the emergency treatment has been completed.

Pre- and Postoperative Care

Parenteral feeding during the pre- and postoperative period of patients requiring major surgery for serious gastrointestinal lesions includes five important

food factors: (1) water, (2) electrolytes, (3) calories, (4) proteins and (5) vitamins. Nutrition, whether enteral or parenteral, must include consideration of all five of these elements because each requires the presence of the others.

The *water* loss of a normal person not subject to exertion or perspiration is 2,500 c.c.[3] Increased insensible water loss occurs in patients postoperatively or in the presence of a fever. An extra 250 c.c. of fluid is required for each degree the temperature is above the normal 98.6° F. Patients with renal impairment[4] and loss of the concentrating mechanism may have an obligatory renal loss which greatly increases their output and requirements. The daily requirement of the average surgical patient without complication has been estimated at 3,000 c.c. to 3,500 c.c.[5]

The average daily *salt* loss is 6-10 gm.[6,7] which can readily be supplied by the salt in one liter of normal physiological saline solution. In certain types of cases in which excessive chlorides have been given in intravenous fluids, the chlorides become trapped in the extracellular tissue spaces. These chloride molecules retain water in a static form in the tissue spaces resulting in edema. While the patient may be receiving adequate fluids, these fluids are static and the patient remains dehydrated and the urine output is diminished[8]. This oliguria may be falsely interpreted as renal failure or kidney shutdown from sulfonamide crystals or some other cause. The proper treatment for this condition is to give fluids without chlorides such as a solution of 10 per cent glucose in distilled water. This water, after circulating through the tissues, will be excreted through the kidneys carrying off some of the excess chlorides. The chloride excretion then permits excretion of the trapped water and diminution of the edema.

The *caloric requirement* of an adult of average age at bed rest has been calculated as 1,200-1,600 calories per day[3]. If insufficient calories are obtained from food intake the body mechanism must draw on stores such as fat. When these stores have been exhausted, the body mechanism must draw on proteins which constitute the building material of muscles and vital organs. A rapid deterioration of tissue results, beginning first in the skeletal muscle structures and last of all in the vital organs. The body store of glycogen is insufficient to supply more than one day's caloric requirement. The metabolism of fat, unless supported by carbohydrates, results in ketosis, and the use of protein to supply calories depletes the essential body tissues. Since fat cannot be given parenterally, the only food elements suitable for administration by this route available at the time of this writing are glucose and protein. When one gives amino acids, or hydrolyzed protein solutions, it is with the purpose of supplying protein for tissue repair and not calories. However, if one does not provide in addition adequate calories, the amino acids will then be de-aminized and used for energy purposes.

Endogenous tissue fat may be expected to supply some of the caloric requirement, yet most patients who require prolonged parenteral feeding have little fatty tissue upon which to draw. One should not require the patient to draw on body reserves to supply more than one quarter of his daily energy requirements or approximately 400 calories. The task of supplying the additional 1,200 calories can

be readily met by administering a solution of 10 per cent glucose in distilled water. While 5 per cent glucose solution is ideal for the treatment of dehydration, its use is not feasible when all the calories must be provided by the parenteral route, since 6,000 c.c. of this solution would be necessary to supply 1,200 calories. There are no disadvantages to careful administration of 10 per cent glucose solution. Glucose can be assimilated by the body mechanism at the rate of 0.5 gm./kilo/hour which is about 35 gm. per hour for the average adult without significant hyperglycemia or glycosuria[9,10]. On our wards we have usually allowed 2½ to 3 hours for each liter of 10 per cent glucose, a rate of flow of about 100 drops per minute.

The *fourth nutritional group* consists of tissue building proteins. For many years little attention was given to protein intake because it was assumed that the body mechanism possessed large stores permitting a person to live for weeks or months without protein intake. Weech and others[11,12] have shown that the proteins of plasma, liver and muscle are rapidly depleted when the intake is insufficient. Brunschwig[13] has emphasized the magnitude of protein loss following operations. The failure of proper wound healing resulting from protein deficiency is well known[14]. The edema which accompanies low serum proteins, particularly in gastrointestinal surgery, may lead to serious complications.

It has been estimated that a person whose protein catabolism is kept minimal by adequate caloric intake requires about 40-60 grams[15] of protein per day. This can be supplied by blood, plasma, albumin, amino acids or casein hydrolysate. Plasma and albumin have the advantage of supplying protein immediately and are of special value in cases where the deficit must be overcome rapidly. We have found casein hydrolysate which is a combination of amino acids and small peptides, an effective substitute for dietary protein.

We have noted very few reactions following parenteral administration of casein hydrolysate. Nausea, vomiting, flushing and vasodilatation when present have been found to be due to too rapid infusion. Fever may occasionally occur and this is usually due to pyrogens in improperly prepared tubing.

We have found that a mixture of 500 c.c. 10 per cent amino acids and 1,000 c.c. 10 per cent glucose makes a very useful solution which contains 50 gm. protein and 100 gm. of glucose. However, stock solution containing 5 per cent amino acids and 10 per cent glucose avoids the possible contamination during preparation, and allows an increase in glucose which will provide 1,400 calories in 3,500 c.c. total fluid (Formula B).

The *fifth group of nutritional elements* consists of the vitamins including thiamin, riboflavin, niacin, ascorbic acid and Vitamin K. The members of the B-complex group are associated with the metabolism of glucose and are important when glucose is given intravenously[16]. The importance of Vitamin C in surgical patients has been well established. The recommended daily dosage for each type is as follows: thiamin 2 to 10 mg.; riboflavin 2 to 5 mg.; niacin 25 to 100 mg.; and ascorbic acid 300 to 1,000 mg.

TABLE I

No.	Pt.	Hosp. No.	Sex	Age	Diagnosis	Operation	Total Protein		Total	Complications
							Adm.	1 Wk. Postop.	Bl. Trans.	
1.	E.B.	1252783	M	76	Carcinoma of rectum	Miles Resection	4.83	5.44	1,000 c.c.	None
2.	E.G.	1256987	M	72	Carcinoma of rectum	Miles Resection	4.81	6.59	1,000 c.c.	Phlebothrombosis Bil.fem.lig.; sl. wnd. infection
3.	S.F.	1256855	F	42	Obst. duodenal ulcer	Subtotal Gastrectomy	5.12	6.43	1,000 c.c.	None
4.	P.D.	1256217	M	38	Obst. duodenal ulcer	Subtotal Gastrectomy	5.39	6.1	1,000 c.c.	None
5.	L.M.	1258835	M	74	Carcinoma of rectum	Miles Resection	5.03	6.32	1,000 c.c.	Slight wound infection
6.	J.D.	1255877	M	45	Gastrojejunal ulcer	Resect. Gastrojejunostomy & Subtotal gastrectomy	5.95	6.54	1,000 c.c.	Atelectasis right base, minor
7.	L.L.	1270070	F	64	Carcinoma stomach	Subtotal gastrectomy & transverse colectomy	5.9	6.4	2,000 c.c. (Adm.RBC 2.87)	None
8.	A.F.	1262049	M	48	Gastric ulcer	Subtotal gastrectomy	7.5	6.85	1,000 c.c.	None
9.	R.S.	1261950	M	56	Duodenal ulcer	Subtotal gastrectomy	6.22	7.27	1,000 c.c.	None
10.	M.G.	1264978	F	44	Bleeding duodenal ulcer	Subtotal gastrectomy	5.71	6.0	1,000 c.c.	None

TABLE I (Continued)

No.	Pt.	Hosp. No.	Sex	Age	Diagnosis	Operation	Total Protein		Total	Complications
							Adm.	1 Wk. Postop.	Bl. Trans.	
11.	F.N.	1269396	M	47	Gastric ulcer	Subtotal gastrectomy	5.05	5.73	1,000 c.c.	None
12.	J.B.	1268069	M	39	Duodenal ulcer	Subtotal gastrectomy	5.44	6.38	1,000 c.c.	None
13.	F.L.	1273804	F	48	Obst. duodenal ulcer	Subtotal gastrectomy	5.78	6.42	1,000 c.c.	Minor wound infection
14.	E.B.	1277875	F	32	Carcinoma of rectum	Miles Resection Total hysterectomy	6.47	7.0	1,000 c.c.	None
15.	L.C.	1279214	F	49	Carcinoma of rectum	Miles Resection	5.92	5.15	1,000 c.c.	None
16.	T.A.	1280386	M	63	Carcinoma of rectum	Miles Resection	5.42	5.9	1,000 c.c.	None
17.	E.P.	1287095	F	56	Carcinoma of sigmoid	Res. & Anast.	5.11	6.2	500 c.c.	None
18.	M.S.	1286482	F	52	Carcinoma of sigmoid	Res. & Anast.	5.74	5.9	1,000 c.c.	None
19.	E.E.	1284995	F	71	Carcinoma of rectum	Miles Resection	6.4	6.0	1,000 c.c.	None
20.	E.H.	1283929	F	74	Cholecystduodenal fistula. Choledocholithiasis. Calculus impacted in Ampulla of Vater	Cholecystectomy choledochotomy duodenotomy	6.2	6.4	500 c.c.	None

While we admit that no routine method of fluid therapy can be applied to every case, yet we feel that one should have a basic formula which can be modified to suit the individual case.

The basic fluid formula should supply:

3,000 - 3,500 c.c. fluid
6 - 10 gm. sodium chloride
300 - 400 gm. glucose (1,200 - 1,600 calories)
40 - 60 gm. available protein
vitamins

These requirements can be supplied with either of the following formulae:

Formula A:

Fluid	*Contents*	*Protein*	*Glucose*	*Calories*
1,500 c.c.	10% glucose in distilled water		150 gm.	600
500 c.c.	10% glucose in saline		50 gm.	200
1,000 c.c.	10% glucose		100 gm.	400
500 c.c.	10% amino acids	50 gm. (for tissue repair)		
3,500 c.c.		50 gm.	300 gm.	1,200

Formula B:

Fluid	*Contents*	*Protein*	*Glucose*	*Calories*
2,000 c.c.	10% glucose in distilled water		200 gm.	800
500 c.c.	10% glucose in saline		50 gm.	200
1,000 c.c.	10% glucose 5% amino acids	50 gm. (for tissue repair)	100 gm.	400
3,500 c.c.		50 gm.	350 gm.	1,400

Either of these formulae will supply the necessary food elements to the average surgical patient who has no abnormal fluid loss such as from Wangensteen suction drainage, diarrhea, or intestinal fistulae. Fluid lost from the gastrointestinal tract must be made up liter for liter by normal saline solution. Additional salt may easily be given by using glucose in saline instead of glucose in distilled water in the above formula. If there is a great salt and fluid loss from the gastrointestinal tract additional salt should be added to replace in amount the salt lost[5].

In surgery of the gastrointestinal tract this method of parenteral feeding may be prolonged until adequate oral intake can be established. As oral intake is increased, the amount of parenteral fluid may be diminished, but it is advisable to continue the protein hydrolysate until complete oral alimentation has been obtained.

The vitamins should not be added to the amino acid mixture, nor should they be given in the same infusion with sulfadiazine. If permanent rubber tubing is used it must be carefully cleaned, by boiling in sodium hydroxide or sodium carbonate to remove pyrogens.

This basic formula has been used on the Fourth Surgical Service at Boston City Hospital in all cases requiring parenteral feeding. We have made a study of the nutrition of these patients over a three-year period and have found that with this method we have been able to provide adequate nutrition for major surgical cases in the critical postoperative period. In each case the basic formula has been modified to supply the individual need as determined by the blood chemistry. Particular attention has been given to the daily serum chloride as a guide to the amount of saline needed. In our cases the incidence of superficial venous thrombosis secondary to intravenous amino acids and 10 per cent glucose has not been a problem. While repeated daily use of the same vein may result in thrombosis, judicious venipuncture and slow infusion will aid in the preservation of veins.

The accompanying table gives the results of this intravenous diet in twenty cases of major abdominal surgery. Serum protein determinations were taken on admission and one week after operation in order to evaluate this formula as a means of adequate parenteral nutrition.

In each of these cases the disease of the gastrointestinal tract was of a debilitating nature requiring surgery of the more major type, both the disease and radical cure requiring great stores of body proteins with little chance of replacement by normal alimentation. It is, therefore, noteworthy that in spite of all this the total serum proteins actually increased in all but three cases on this regime.

Surgical Complications

1. Intestinal Obstruction:—The patient with intestinal obstruction is suffering from dehydration, hemoconcentration and marked salt loss. The hematocrit and plasma protein determinations are markedly elevated. The needed salt and water can usually be supplied by 2-3 liters of 5 per cent glucose in saline. The quantity and type of parenteral fluid needed may be determined by specific gravity and amount of urine, serum chloride, hematocrit and total protein. As soon as acute loss is corrected the patient may be placed on a basic formula plus the amount of normal saline solution equal to Levine or Miller-Abbott tube drainage as suggested by Coller[8].

2. Acidosis Alkalosis and Hypochloremia:—Abnormal loss of the gastrointestinal secretions produces disturbance of the acid-base balance. In the case of vomiting, there is a loss of chlorides with resultant alkalosis, while in the case of diarrhea there is loss of alkaline intestinal secretions resulting in acidosis. The loss of large amounts of alkaline pancreatic or biliary secretions will also lead to acidosis.

In all cases of vomiting it is of the utmost importance to evaluate the chemical picture with particular emphasis being placed on the state of hydration of the patient. Evidence of *alkalosis* includes muscle hypertonicity as shown by: exaggerated reflexes, Trousseau and Chvostek signs, and the presence of deep and exaggerated respirations. An accurate estimate of the degree of alkalosis may be made by determination of the CO_2 combining power and serum chloride. The treatment of alkalosis consists of 2-4 liters of normal saline and occasionally even 2

per cent saline solution. Frequent laboratory determinations of CO_2 combining power and serum chlorides should be made.

Acidosis occurs in diarrhea, starvation and diabetes. The degree of hyperpnea and the decreased CO_2 combining power provide an estimate of the severity of the condition. For treatment, sodium R. Lactate 1/6 molar solution is the solution of choice. This substance changes rapidly into bicarbonate and glucose which immediately compensates for acidosis[18]. The recommended dosage is 125 c.c. for each volume per cent that plasma CO_2 is less than 55, in a 60 kilo adult[17].

Hypochloremia must be carefully watched for in patients on Wangensteen suction or with biliary or intestinal fistulae. This state of low serum chloride may appear insidiously with an initial complaint of abdominal cramps, followed by prostration, rapid pulse, falling blood pressure and a state of shock. If this condition is not quickly recognized and treated with saline, a fatality may result. A formula for estimating the amount of chloride required has been suggested by Coller[7]. He recommends 0.5 gm. NaCl/kilo for every 100 mg. serum chloride deficit.

3. Peritonitis:—Peritonitis can readily produce clinical shock. The chemistry disturbance simulates that of a severe burn case with loss of protein and water. The hematocrit is elevated and the total protein lowered. Transfusion of whole blood or plasma should be used freely as indicated to restore acute protein loss as quickly as possible following which amino acids may be given, to supply additional protein. This may be done by using 2,000 c.c. of glucose and amino acids daily. The above formula can be changed to meet this need by adding 500 c.c. of 10 per cent amino acids to 1,500 c.c. 10 per cent glucose in distilled water. The associated dehydration and acidosis must be managed according to individual needs. If peritonitis occurs as a hospital complication in a patient who has received adequate fluids, the dehydration and acidosis should not be a problem. Patients who enter the hospital with a general peritonitis require emergency treatment with sufficient whole blood, plasma and fluids including saline and sodium R lactate to restore electrolytes to normal.

Summary and Conclusions

1. Traumatic shock requires replacement in kind and amount the total fluid loss, i.e., whole blood and plasma.

2. Adequate and expeditious therapy of the seriously injured requires a "shock" team.

3. Total parenteral therapy requires accurate estimation of the patient's needs in terms of water, electrolytes, calories, proteins, and vitamins.

4. A combination of 10 per cent glucose and casein hydrolysate is proposed in a basic formula.

5. A series of 20 cases with serious gastrointestinal lesions requiring major abdominal surgery which received this regime are tabulated. All, with minor exceptions, were nutritionally improved as indicated by increase in the serum proteins.

6. Intestinal obstruction, chemical imbalance and peritonitis are discussed in relation to fluid therapy.

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PRIMARY TORSION OF THE OMENTUM*

JULIAN A. STERLING, M.D., M.Sc. (Med.), F.A.C.S.†
and
RALPH GOLDSMITH, M.D., F.A.C.S.††
Philadelphia, Pa.

Introduction

Omental torsion may be considered to be a twisting of the omentum sufficient to compromise its distal circulation. It is of interest because it produces abdominal pain, and frequently a palpable mass.

The primary type of torsion of the omentum is rare, and is due solely to a spontaneous twist. It was first reported by Eitel[5] in 1899. There has been but one mortality in the seventy patients reported[1] through 1946. This low mortality is not surprising, since the omentum is not a vital organ.

Secondary torsion is more common than the primary type. It is found very frequently in association with inguinal and femoral herniae; many hundreds of such cases[8] have been reported. Secondary torsion of the omentum is also found contiguous to primary disease of any abdominal viscus.

TABLE I
Report of Questionnaire on Primary Omental Torsion

City	Atlanta	Brooklyn	Denver	San Diego	Phila.	Total
Number of questionnaires	31	85	45	26	106	293
Number of replies	14	17	14	14	41	100
Zero cases	11	8	12	12	21	64
Positive report	*3*	*9**	*2*	*2*	*20*	*36*
Number of cases Reported	7	18*	2	2	46	75

(*) includes three cases previously reported.

Statistical Survey

Since so few cases of primary omental torsion had been reported, we were curious regarding the actual frequency of unrecorded cases. Therefore, a survey was conducted in October, 1949, among general surgeons who were members of the American College of Surgeons[16]. Two hundred ninety-three questionnaires were sent to surgeons in Atlanta, Ga.; Brooklyn, N. Y.; Denver, Colo.; Philadelphia, Pa. and San Diego, Calif. One hundred replies were received.

Thirty-six surgeons reported 75 cases of primary omental torsion. Three of these cases had previously been reported. In this survey, then, 72 unreported cases appeared among 100 surgeons who replied to the questionnaire (Table I).

*From the Surgical Service of the Jewish Hospital, Philadelphia 41, Penna.
†Assistant Surgeon.
††Chief Attending Surgeon.

In addition, 15 surgeons stated that they had seen more than 81 patients with secondary omental torsion. Forty-nine (60 per cent) of these cases were found to be associated with inguinal herniae (Table II).

It is apparent from the above that the incidence of this condition is greater than had been believed.

Symptomatology

The clinical manifestations[2] of primary omental torsion are:

(a) abdominal pain—dull, constant and subumbilical.
(b) nausea, often without vomiting.
(c) irregularity in colon function.
(d) the appearance of a palpable mass (abdominal or pelvic).
(e) duration of 2-3 days.

TABLE II

Report of Questionnaire on Secondary Omental Torsion

City	Atlanta	Brooklyn	Denver	San Diego	Phila.	Total
SURGEONS REPORTING CASES	4	3	3	1	4	15
NUMBER OF CASES	46+*	10	5	8+	8+	81+
Inguinal Hernia	35+	5	4	0	5+	49+
Femoral Hernia	7	1	1	8+	1	18+
Other Hernia	2	2	0	0	2	6
Other Causes	2	2	0	0	4	8

(*) JCR reported 40 + cases.

The most frequent preoperative diagnosis is acute appendicitis[3]. Any intra-abdominal crisis, however, can be simulated by this condition. The diagnosis of primary omental torsion can be made preoperatively only if it be included among all possible causes for a surgical abdomen. Surgical therapy is usually indicated for patients with abdominal pain, nausea, and signs of peritoneal inflammation, hence, the specific preoperative diagnosis of primary omental torsion is almost entirely of academic interest.

Case Reports

In the past decade (1940-9) three cases of primary omental torsion were observed at the Jewish Hospital, Philadelphia. More than 40 cases of secondary omental torsion were reviewed during that same period at the Jewish Hospital, but this latter study is not reported.

Case 1:—#231587, M. B., white female, aged 40, admitted January 13, 1947 (Service Dr. I. Forman). Complaint on admission was constant, sharp, lower abdominal pain of one week's duration. Appendectomy had been done previously. On pelvic examination, a tender mass was palpated involving the right adnexae. Preoperative diagnosis was: twisted ovarian cyst. Operation was performed on January 14, 1947. A mass of twisted and necrotic omentum was found adherent

to the dome of the bladder and to the right tube and ovary. Adnexae and uterus were normal grossly and histologically. Exploration of the abdomen was otherwise normal. The omentum was hemorrhagic and necrotic. Microscopic examination (Fig. 1) revealed an arterial thrombus, edema and cellular infiltration.

Case 2:—#246429, F. T., white female, aged 49, admitted April 27, 1948 (Service Dr. N. S. Rothschild). Complaints on admission were: dull, right lower quadrant pain of 48 hours duration, and nausea without vomiting. Preoperative diagnosis was: acute appendicitis. Operation was performed April 27, 1948. The appendix was normal. A gangrenous segment of omentum was found adherent to a normal ascending colon. The mass was approximately 3 cm. in diameter. Gross examination of the excised omentum revealed infarction. Under the microscope, (Fig. 2), red blood cell extravasation was seen.

Case 3:—#263692, J. W., white male, aged 57, admitted September 25, 1949. Complaints on admission were: Lower right and midabdominal pain of five days'

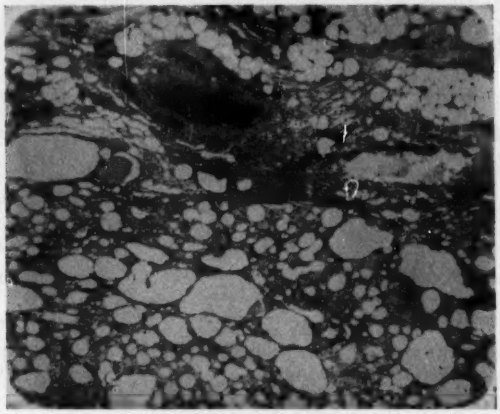

Fig. 1—(Case 1)—Hemorrhage, edema and arterial thrombosis in greater omentum. (X 60)

duration, anorexia and nausea without vomiting, and watery diarrhea secondary to saline purgation. Pain had started while the patient was working as drill-press operator. He was seen 24 hours after onset of pain and nausea. T. P. R. were normal. There was abdominal tenderness above McBurney's point with moderate rigidity of the right rectus. Rebound phenomena were referred to the right lower quadrant. Peristalsis was normal. Rectal examination was normal. There were no herniae. There were 17,500 white cells with 87 per cent polys. A diagnosis of acute appendicitis was made and operation advised. This was refused and the patient was returned to his family doctor.

Three days later his doctor referred him for reexamination, this time with a tentative diagnosis of carcinoma of the ascending colon. Pain and anorexia had persisted; there was no vomiting. Tenderness was present to the right of the umbilicus. An egg-shaped mass approximately 10 cm. in diameter was palpated in the right lower quadrant extending upward to the right of the umbilicus. Mod-

erate right rectus rigidity was present. Rebound phenomena were generalized. Peristalsis was normal. Temperature was 98.4°, pulse rate 98 and respirations 22. There were 10,100 leucocytes, 64 per cent of which were polymorphonuclears. Preoperative diagnosis was perforated appendicitis with local peritonitis and abscess formation. Examination under spinal anesthesia confirmed the presence of the mass, which, however, was not in the usual location of an appendiceal abscess.

A right midabdominal muscle-splitting incision was made. Abdominal muscles and peritoneum were edematous. About three ounces of sanguineous peritoneal fluid was present (culture was contaminated by Staph. albus.). The appendix was normal; it was removed. A mass of omentum was found adherent to the ascending colon and the anterior parietal peritoneum. It was easily mobilised. The right side of the omentum had been rotated several times, was hemorrhagic and necrotic, and measured 8 by 6 by 3 centimeters. The easiest method for ex-

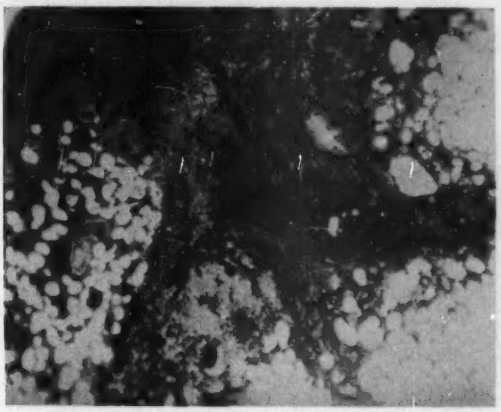

Fig. 2—(Case 2)—Infarction of Omentum. (X 12)

cision of this mass was to detach the omentum along its cleavage line to the transverse colon at the hepatic flexure. Suture-ligatures were placed in healthy omentum laterally and proximally. The twisted omentum was removed without derotation. Exploration of the abdomen was otherwise negative.

Gross examination revealed infarction and necrosis of the omentum. Microscopic examination (Fig. 3) showed necrosis of the omentum, cellular infiltration and an unorganized venous thrombus.

All three patients had an uneventful convalescence and have since been symptom-free.

Discussion

The most familiar examples of torsion of intraabdominal organs are those affecting cystic and solid tumors of the ovary[10]. An intraabdominal organ which is attached to a mesentery or a stalk is particularly subject to acute torsion[12]. In the intestinal tract torsion can occur as a volvulus, and as such can produce intestinal obstruction.

Axial rotation has a true mechanical basis founded on the principle of the couple. This couple can be provided by two viscera, contracting against each other. It can also be produced by one viscus and a resistance such as the sacrum. There is a natural limit on rotation because of the arterial circulation and the sheathing tissues[6].

Torsion is significant clinically when circulatory embarrassment occurs. Partial torsion, or incomplete vascular obstruction of the omentum, may be asymptomatic. This is possible, since there are no efferent nerve fibres of the omentum[1], and clinical symptoms are usually produced by secondary involvement of the mesenteries, serosal surfaces, or peritoneum. Under some circumstances a twisted omentum may spontaneously derotate. At operation, the twist cannot be found, and only a bruised and hemorrhagic area is found in the omentum. This probably represents the so-called "omental apoplexy"[7].

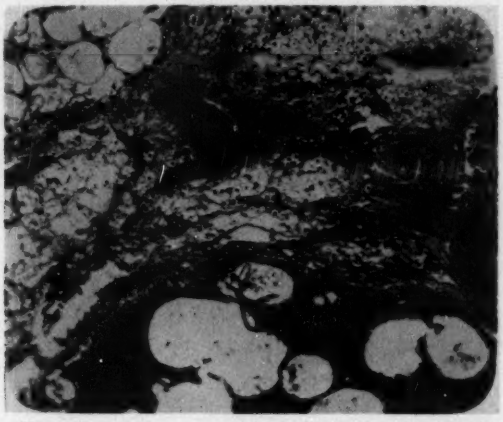

Fig. 3—(Case 3)—Hemorrhage and an unorganized venous thrombosis in the omentum. (X 12)

In the early stages of omental torsion there is vascular engorgement and a perivascular cellular infiltration with edema (Fig. 3). Later, there is infarction with necrosis (Fig. 4). It is at this stage that primary omental torsion is usually subject to surgery. It is possible for the twisted omentum to atrophy or become fibrotic. This is seen frequently in patients who have omentum strangulated in a hernia sac for a long time.

Omental torsion occurs most frequently on the right. Perhaps this is because the right side of the omentum is longer, larger, heavier, and more mobile than the left[9].

The omentum has no inherent motion. There is no muscle tissue in omentum, except for that present in the arteries. Omental motion does not occur in response to inflammation, nor can chemotaxis provoke movement[14]. Hence, any motion of the omentum is due to the motion of tissues and organs in contact with it. In discussing certain phases of adhesion formation in the abdominal cavity of rats, Rhoads[13] stated as his impression that the omentum became attached to the

sticky area exuding from any area of bowel wall injury being carried to these areas as a result of random movements of respiration and peristalsis. There was a tendency for the omentum to surround the area of injury and to be held there by the adhesive properties of the exudate during the first stage of formation of an adhesion.

There are three theories regarding the production of omental torsion.

(1) Intestinal motion: The most frequent source of motion in the peritoneal cavity is intestinal activity. It is postulated that peristalsis or mass movement changes the normal flatness of the omentum to a coil, which continues to roll[6].

(2) Vascular: Veins are longer and larger than the arteries. This permits motion around the artery as the axis[11,15]. Rotation greater than 90 degrees may obstruct the venous circulation. It may be that primary vascular disease of the omentum is a precursor to primary omental torsion. Our studies do not confirm this, for the vessels have all been normal except for the presence of thrombosis.

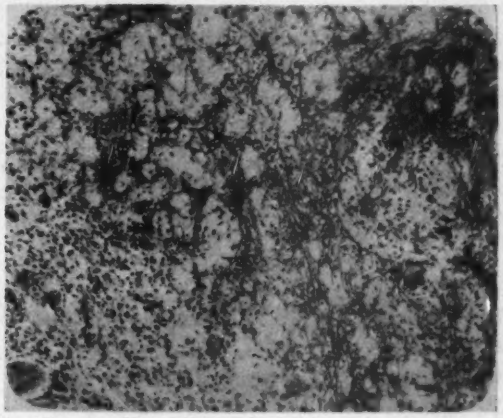

Fig. 4—(Case 3)—Hemorrhage and necrosis in greater omentum. (X 60)

We have not found any evidence of arterial or arteriolar sclerosis as a precipitating mechanism in omental torsion.

Because of the existence of venous thrombi, which are not organized, it is unwise to "derotate" or otherwise manipulate the pedicle of the twisted omentum. The surgical maneuver of choice is suture-ligature of small segments in normal tissue.

(3) External trauma: A force introduced from outside the abdomen may produce an abnormal twist of the omentum. It has been suggested that a direct blow, violent exercise, or abnormal body movement can impart an axial twist to a mass of omentum, which continues to rotate because of inertia[8,9].

The preoperative diagnosis of primary torsion of the omentum is both difficult and unnecessary. It is important, however, that the diagnosis of omental disease be made at the time of laparotomy in the presence of a suspicious abdominal mass, or sanguineous peritoneal exudate.

Summary

(1) Three cases of primary omental torsion with full recovery are presented from the Jewish Hospital during 1940-9.

Seventy-two unreported cases were tabulated as a result of a survey among 293 surgeons in 5 urban communities.

(2) The diagnosis of primary omental torsion cannot be made preoperatively except if it be included in an exhaustive list of causes for the "Acute surgical abdomen".

It may be significant that the patient with primary omental torsion has:

(a) abdominal pain of several days' duration

(b) nausea without vomiting

(c) a mass in the right midabdomen

(3) Theories concerning the etiology of omental torsion have been reviewed. Factors of (a) intestinal movement, (b) specific blood vessel anatomy, and (c) external trauma were listed.

(4) It has been emphasized that the twisted portion of the omentum should not be derotated, and that excision should be accomplished within a normal segment, because of the danger of liberating a thrombus.

Conclusion

Primary torsion of the omentum probably occurs with greater frequency than has been previously believed. A total of 145 cases are now known.

The authors thank Drs. Forman and Rothschild for permission to review their cases.

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ACUTE PERFORATION OF GASTRIC AND DUODENAL ULCERS*†

A Report of 600 Cases

JOHN B. O'DONOGHUE, M.D., F.A.C.S., F.I.C.S.
MAURICE B. JACOBS, M.D., F.A.C.S., F.I.C.S.
and
JOHN B. O'DONOGHUE, JR., M.D.
Chicago, Ill.

For over three hundred years the medical literature of the world has frequently stressed the catastrophe of the perforation of a gastrointestinal ulcer. The accumulation of the vast literature on this subject testifies to its recognized importance and the challenge it has given to medical science.

Previous to the turn of the century Moynihan and Mikulicz reported mortality rates in perforated ulcer to be 91.1 per cent and 68 per cent respectively. In the first two decades of the present century the average mortality rate was about 40 per cent. During the third decade 20 per cent to 30 per cent was the average, notwithstanding the great advances in surgical technic and the greater knowledge of the physiology of the stomach. I am happy to be able to show you a great improvement in recent years.

Since the first report in 1671 of the finding at postmortem of a gastrointestinal perforation, and in the succeeding 279 years, many descriptive reports of the condition have been made by de Muraldo, 1688; Christopher Rawlinson before the Royal Society in 1729; Hamburger, 1747; Travers and Compton, 1817; monographs by Muller, 1860; and Kraus, 1865. In 1875, O'Hara presented the first report of a perforated duodenal ulcer before the Philadelphia Pathologic Society. The first operation for a perforated ulcer was performed by Mikulicz in 1880, the patient dying of shock in three hours. The first successful operation was performed by Huessner in 1892. Two years later, 1894, Otterton operated the first successful case in America. In 1892 Hall collected six cases in which spontaneous recovery occurred.

In the ensuing years the surgical treatment of perforated ulcer became more common, and I would like to stress that point today, namely, that there is only one treatment for perforated ulcer, and that is surgical. I have purposely called your attention to the six cases collected by Hall in 1892, in which spontaneous recovery occurred, and further, would like to stress the alarming frequence with which reports are appearing in the American and English medical literature of recovery from perforated gastrointestinal ulceration that has been treated conservatively. I will treat this subject more completely when I come to the discussion

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†From the Department of Surgery of the Stritch School of Medicine, Loyola University, Cook County Graduate School of Medicine.
Hektoen Institute of Medical Research of Cook County Hospital, Chicago, Ill.
Department of Surgery, Cook County Hospital, Chicago, Ill.

of treatment. The surgical treatment has been influenced in each decade by the evolution of surgery; in the antiseptic era, the aseptic era, the period when Lord Moynihan's teachings of the modern principles of gastrointestinal surgery were accepted, and since then by the great teachers in Europe and America; among them, Finsterer, Stipa, Bruner, Judine, Murphy, the Mayos, Dever, Allen, Crile, Lahey, Alton Ochsner, DeBakey, as well as many others.

Notwithstanding the vast army of investigators and their endless studies, this catastrophe remains one of the most perplexing to medical science today; is the number one surgical emergency and stands in twelfth place as the cause of death.

Our interest in this subject dates back a quarter of a century and I am prompted to bring to your consideration our studies and conclusions obtained from six hundred cases of gastrointestinal perforation because of the information and other certain pertinent facts that are evident. These cases have all been proven at operation or at postmortem. All cases of subacute ulcers, penetrating and the forme fruste type have been excluded.

We believe that we enjoy an unique opportunity in that Cook County Hospital in Chicago is the only charitable type of general institution that is entirely tax supported and receives the majority of emergencies that arise in that large metropolis. Other large cities having possibly a greater number of emergencies provide for their treatment among several institutions. Accordingly, a large number of cases are observed in a relatively short period of time, the treatment instituted is similar and the operative and postoperative treatment carried out by the same group of surgeons are the same.

The material for our study considers 600 patients who have been classified into three groups. The first group of 200 cases were treated during 1935 through 1937. This represents the prechemotherapeutic and preantibiotic era; the second group of 200 cases were treated a decade later during 1944 through 1946. This period was marked by the extensive use of the sulfonamides. The last group of 200 patients were treated during the interim of January, 1948 through May 11, 1950 wherein antibiotics were the principal adjunct of treatment.

The medical literature from the clinics through the world show an increased incidence of perforated peptic ulcer everywhere. DeBakey, reporting from the Charity Hospital in New Orleans for the years 1929 to 1938, shows that, based on the number of admissions, the number of ulcer cases was 0.478 per cent and the perforated ulcers was 0.038 per cent. In our series of 1935-1937, the percentage of ulcer cases admitted was 0.71 and the perforations 0.0365. In the 1944-1946 series the percentage of ulcers admitted was 0.66 and the perforation 0.062. In the series of 1948 to 1950 the percentage of ulcers admitted was 0.83 and the perforations 0.63. The results in our series of cases, coinciding with the statistics from all over the world, show that the incidence of peptic gastrointestinal ulceration is rapidly increased and that there is a disproportionately greater increase in the percentage of perforations. This corresponds with reports by Stipa, Shawan, DeBakey and others and we believe that the change in the pattern of life, with its anxiety and tension and poorly prepared food, as well as hasty eating are important factors in

causing the increased incidence of ulcer. The use of tobacco and alcohol certainly play a very important part.

As one reads the history of ulcer we cannot help being impressed by the change in the incidence of ulcer in men and women. There are any number of reports showing the high incidence in the female in the latter part of the last century and up to about 1920. Since then ruptured ulcer has occurred in 98 per cent to 99 per cent in men until in the last three years we are again seeing an increased incidence in women. In the three groups the percentage has been 1.5, 2 and 5 respectively. Whether or not women with their gain of suffrage since 1917 are now beginning to suffer from the anxiety and tension of modern living remains to be seen. It is common knowledge that they are using tobacco and alcohol almost as frequently as men.

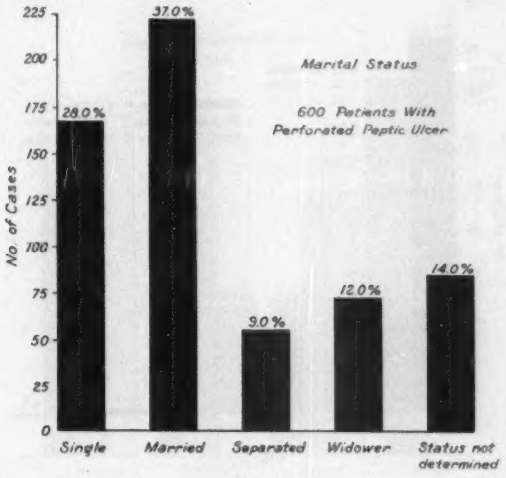

Fig. 1

In the three groups studied, all show that perforation occurs much more frequently in married men, again bringing to our attention the psychosomatic factor or as Dr. Ivy likes to call it, the emotogenic factor.

In studying peptic ulcer in the different races we have wanted to say in the past that it occurred more frequently in the white man, especially in countries where there was some fault in diet or that "he labored under the sweat of his brow". There are many fallacies in this. It has been shown by reports from many countries that given the Negro under the same undesirable circumstances as the white underprivileged individual, the incidence of ulcer is the same and in our series we are able to show that in the last two series the incidence is not only the same but the Negro develops peptic ulceration and perforation ten years earlier than the white man. While it is true that those in strenuous mental enterprise, as executives, professional persons, and people of great responsibility tend to develop

peptic ulcer, it is nevertheless a fact that the incidence of peptic ulcer and perforation are far more frequent in the poor, ignorant, underprivileged group whose mode of life denies them the personal habits of oral and dental hygiene, as well as a well-prepared, clean and balanced diet.

To say that the seasons of the year have much to do with the occurrence has not been brought out by our studies. Twenty years ago we felt that ulcer occurred more frequently in the spring and fall. Petersen has tried to prove through the years that barometric pressure was a factor. In our three series there has been no great variation and during the years 1948-1950 there has been least variation. The spring showing the highest incidence.

In our series the age at perforation ranges from 3 days to 83 years old. The age incidence of ulcer has always been interesting and our experience coincides

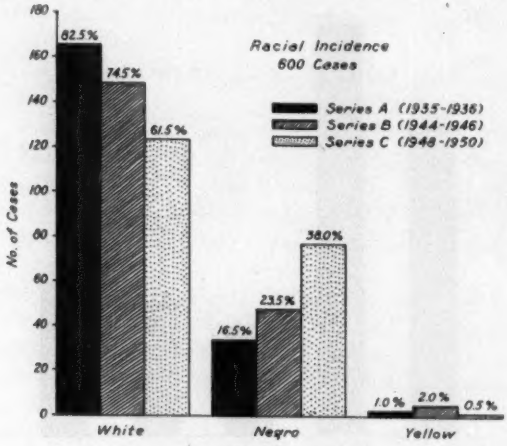

Fig. 2

with that of Thelander, Stern, Schmidt, Kennedy, Meyer, Brams, DeBakey and many others. We have noted, as they have, that perforated ulcer like peptic ulcer occurs in the third, fourth and fifth decade, but recently we have been forced to realize that perforated ulcer is occurring with increasing frequency in the later decades of life. And we see in these cases of perforation occurring in the sixth, seventh and eighth decade a picture that is truly as similar as the one occurring in the earlier decades. This fact is true in all surgical diseases and is especially true in peripheral vascular disease. There is no end to the examples of angiospasm occurring in older males which respond to sympathetic block who formerly were victims of gangrene and lost their life with their leg. We cannot help but believe that there is a comparable state in peripheral vascular phenomena and the pathogenesis of perforated peptic ulcer.

One of the very interesting cases in the 1948-1950 series is that of a male infant delivered at term who lived three days and died of a perforated duodenal

ulcer. Postmortem examination showed no evidence of a brain injury although the labor was long and difficult. It is common knowledge that trauma to the diencephalon, or the optic thalamus or floor of the third ventricle or any intracranial injury may predispose one to gastrointestinal ulceration.

That man's longevity is greater than three score and ten has been shown in many ways. Among them is the fact that peptic ulcer as well as perforation is on the increase in the geriatric years. Our second and third series show a marked increase in the seventh and eighth decade. It is interesting to note that in the 1935-1937 series only 7 patients were over 60 years old, while in the later series 1944 to 1946, and 1948 to 1950, there were 30 patients over 60 years of age in each series. Hence 15 per cent over 60 years in each series. DeBakey points out that in a collected series of 6,875 cases in the years 1929 to 1938 only 7.46 per cent were over 60 years of age.

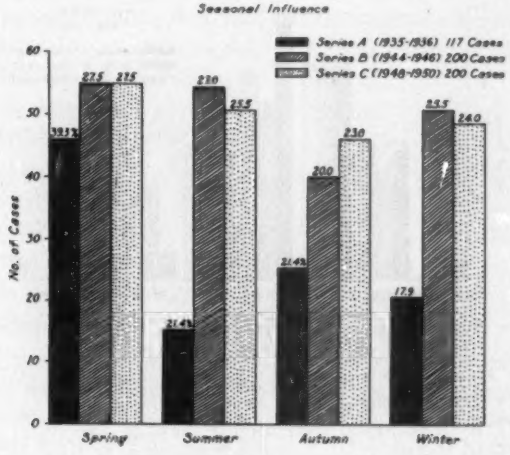

Fig. 3

In studying our series we could find nothing that pointed to the time of perforation in relation to the patient's activity as indicative of the pathology. The only conclusion that might be drawn is that the use of alcohol and tobacco may predispose one to perforation. That some patients (8 per cent in our series) perforate during sleep, is a fact easily explained by the hypermotility, hypersecretion and hyperacidity of the stomach during hours of sleep as well as wakefulness, and again suggests that the acid theory is not adequate in itself to explain the cause of ulcer. We would like to call attention to the fact that the great majority of perforations occur in the area of the gastrointestinal tract occupied by the neutral glands that produce the buffer solutions or the inhibitory "stuff". In the duodenum we find the majority of perforations in the first inch to inch and a half of the anterior or upper surface, certainly that part of the duodenum with a

questionable blood supply, also the part that has most to do with the production of internal secretions.

In discussing the subject of etiology I do so with trepidation of being misunderstood. In the past it was sufficient to say that we do not understand the etiology of ulcer and let it go at that. Today such a statement to my mind lacks certain elements of truth. With your indulgence I would like to point out certain proven accepted facts, namely:

1. That hydrochloric acid is produced by the parietal cells located to the greatest extent in the fundus of the stomach.

2. That secretion of these substances is initiated by

(a) Cephalic stimulation by way of the vagus caused by conscious or unconscious cerebral stimuli associated with food or emotion and is active even in sleep.

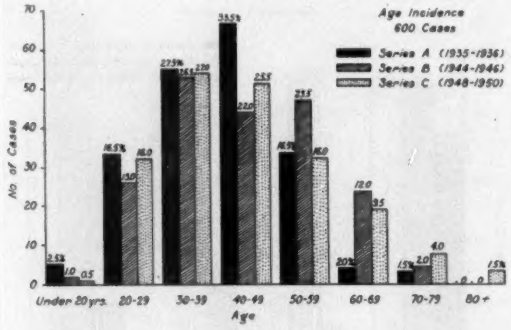

Fig. 4

(b) Gastric stimulation

(1) Through the intrinsic nerves of the stomach
(2) By mechanical distention of food or gas
(3) By chemical action of the digestion of food
(4) Hormonal through the action of gastrin liberated by the antral mucosa.

(c) Intestinal stimulation indirectly by food, enzymes, hormones and intestinal peristaltic action.

3. In every case of ulcer diathesis there is, throughout the twenty-four hours, a hyperacidity, hypersecretion and hypermotility of the stomach in practically every case especially of duodenal ulcer and in the majority of gastric ulcers.

4. Glands are present in the pyloric and cardiac region that have no parietal cells and secrete only an alkaline mucinous secretion that contains mucin, neutral chlorides, sodium bicarbonate, potassium and calcium. The acid-alkaline components of gastric secretion vary inversely in their proportion, one rising while the other falls, possibly controlled by a depressor "stuff" liberated in the pyloric region.

5. It is our humble opinion that in all cases of perforated ulcer there is a vascular factor producing an angiospasm thereby lowering the local resistance of the tissue and then permitting peptic digestion to occur, hence the perforation. Such a group of circumstances could occur by:

(1) Bombarding the vagi nerves with impulses.

(2) Setting free an abnormal amount of histamine or some other product such as acetylcholine causing vasospasm.

(3) Abnormal contractions of the muscularis mucosa in an area supplied by end arteries and in an area of lowered vascular volume.

(4) A depression of the regulating mechanism of the stomach in one suffering from angiospastic disease, or the ulcer diathesis.

(5) Depression of the function of the organs producing the neutralizing secretion of the upper small intestine, namely Brunner's glands secretion, decreased succus entericus, decreased pancreatic juice, decreased bile.

(6) Mechanical trauma within the stomach or duodenum causing irritations and inflammations.

(7) Low healing power as occurs in individuals with a low blood protein or low or absent Vitamin C.

(8) That vascular emboli can occur cannot be denied, as shown by the work of Rosenow.

(9) Chemical irritation by a hyperacidity.

(10) Infection as from teeth or oral cavity or food.

(11) Angiospasm due to hormonal or nervous imbalance.

(12) Neurologic, psychosomatic or emotogenic as Dr. Ivy points out.

Pathology:—The gross pathology may be one of two varieties, viz.:

(1) A soft lesion, usually small, varying from one millimeter to one centimeter in diameter, showing marked evidence of an attempt to seal itself off with mucus and fibrinous exudate which acts as a plug and frequently is supported by a tag of the fatty omentum. Here we note blanching of the edge of the ulcer with freezing or agglutination of all the layers of the viscus, hence obstruction to all blood and lymph flow in the area involved with marked edema of the tissues peripheral to the ulcerative area. This type offers the best prognosis when operated early.

(2) Secondly, the callous lesion which is larger, often by two centimeters, is punched out with sharp, red, necrotic edges and on microscopic examination shows evidence of degenerative arterial endarteritis. This lesion most frequently occurs in the stomach, and is usually seen in individuals past 40 years of age.

One reads in the many papers written on perforated peptic ulcer of the various locations of the perforations. We have been impressed by the fact that 93 per cent to 95 per cent are within one inch proximal or distal to the pyloric sphincter. That only a very small percentage of ulcers occur on the lesser curvature

above the incisura angularis, and less frequently on the greater curvature and the anterior or posterior wall. Practically all of the perforations in the stomach of a peptic ulcer character occur in the antral zone where the glands that produce the neutral secretion are located. A very important fact is that 69 per cent give a positive history of stomach trouble and 29 per cent give no history of stomach distress, 2 per cent did not mention any stomach complaint in their history.

Clinical Manifestations:—The picture presented by a patient suffering from an acute perforated ulcer is dramatic. Moynihan has described it aptly as an "agony suffered almost beyond belief", written on every line of a face that speaks of torture. The face is pale, haggard and watchful. The brow and temples are bathed in sweat, the hair soaked. The patient struggles for breath in short, panting respirations which are wholly costal, for the diaphragm, being an abdominal muscle, is fixed. Words spoken are jerked out in expiration only, every syllable is part of a deep moan. What strikes every onlooker is that the body of the patient is rigid and motionless, no slightest movement dare be attempted. If an endeavor

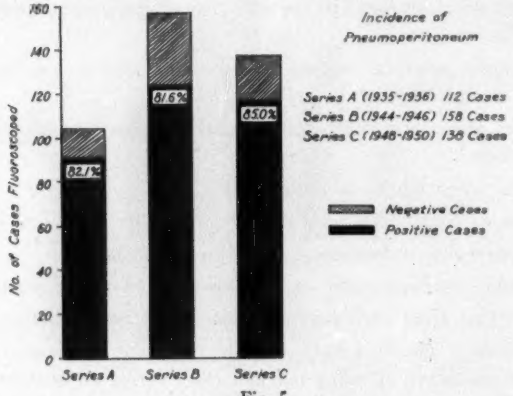

Fig. 5

is made to touch the abdomen, the patient's hands are at once lifted in protest and protection. The pain which is the signal of rupture is caused by pylorospasm and the chemical irritation of the digestive juices and bile coming into contact with the peritoneum.

Vomiting occurred in 60 per cent, it varied from one to ten times and in three cases the vomitus consisted of frank blood. In 30 per cent vomiting did not occur at any time. Nausea was present in only 8 patients, while in 92 cases no mention of either of these symptoms was made in the history. Reference of pain to one or both shoulders was volunteered by 15 patients, however the majority of the patients were not closely questioned on this point.

Physical Findings:—These consisted of abdominal tenderness and rigidity, the former being most marked in the upper abdomen. The rigidity is characteristic and best described as being "board-like". The temperature and pulse rate in the early or primary stage of perforation usually are not elevated. Generally they are normal

or subnormal. One of the most constant findings in acute perforation of a gastroduodenal ulcer is the change in intestinal peristalsis. There is a paucity of literature on abdominal auscultation which is surprising inasmuch as the "silent abdomen" is pathognomonic of peritonitis. The late John B. Murphy used to refer to the abdomen in acute diffuse peritonitis as being "silent as the grave". In over 95 per cent of this series the peristaltic sounds were absent. Hemorrhage was associated with the perforation in 27 patients or 13.5 per cent.

Examination of the blood was performed and was found to vary from 3,250 to 32,200 leucocytes with an average of 15,000 per cubic millimeter, together with a preponderance of polymorphonuclear cells. In ten of the fatal cases the leucocyte count upon admission was below 9,200 per cubic millimeter.

Fluoroscopy:—Fluoroscopic examination, with the patient in the upright and lateral decubitus positions, was done in all cases for evidence of spontaneous pneumoperitoneum. This was positive in 86 per cent of cases. In 14 per cent no free air was present. The absence of spontaneous pneumoperitoneum in cases of acute perforation of a peptic ulcer has been ascribed as being due to variable factors, among which may be mentioned the absence of air in the gastric viscus at the time of perforation, the immediate sealing of the perforation by mucus or food particles or to the presence of perigastric adhesions which prevent the escape of gas into the free peritoneal cavity.

The amount of air that must escape in order to be visible has been investigated by Paine and Rigler. They found a quantity as little as five cubic centimeters in the right subphrenic space could be visualized fluoroscopically or on the x-ray film. Ten cubic centimeters can be seen regularly without difficulty.

In addition to fluoroscopic examination for the demonstration of spontaneous pneumoperitoneum, a flat plate should be taken in the upright and in the left lateral decubitus positions, since minimal free air may be missed on fluoroscopic examination and be shown on the x-ray.

In the differential diagnosis the first conditions to be eliminated are acute appendicitis and acute coronary occlusion; in neither of these conditions are the symptoms and their relation to one another, as I have pointed out under the diagnosis of acute intestinal perforation, demonstrated as they are in this disease. In appendicitis the relation of symptoms and findings are important and not of the severe grade that one finds in perforated gastrointestinal ulceration. The next most frequent conditions thought of are acute pancreatitis, showing an early elevation of the blood amylase; acute cholecystitis, with characteristic relation of symptoms and reference of pain; and lastly acute intestinal obstruction which usually has an antecedent lesion, absence of the abdominal rigidity and abdominal auscultatory silence, the other less frequent conditions to be eliminated are mesenteric thrombosis, intussusception, pneumonia, ruptured ectopic pregnancy, lead colic, ptomaine poisoning, renal colic, twisted ovarian cyst, acute alcoholism, diaphragmatic pleurisy, rupture of tuberculous, or typhoid ulcer and gastric crisis.

The prognosis depends primarily on whether the patient is operated in the first six hours. In our last series we show no mortality in 56 cases operated within

the six hour period. A fatal outcome is increased by procrastination or entertaining for a moment conservative management in cases ruptured within 24 hours when seen. The only cases that should be treated conservatively are those in the group that have ruptured over 24 hours when seen. Any one with experience in this disease knows that there is no way of estimating the size or location of the perforation or whether it is malignant or benign. There are no tests whereby we can tell the amount or reaction of the tissue to spillage, nor are we able to tell when a perforation has been sealed or whether it will leak and reopen again. Doctors Zollinger, Allen and others have made a plea against waiting in the treatment of perforated ulcers and I think that the figures, as shown in our series of 1935-1937, with a mortality of 17.85 per cent in cases operated within 6 hours and the fact that there were no deaths in the series of 1948-1950 when operation was performed within

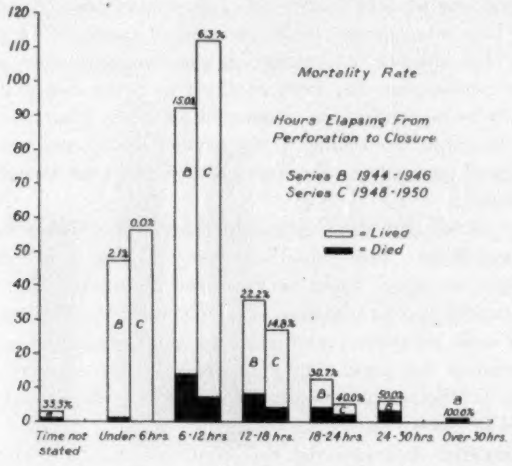

Fig. 6

the 6 hour period, tells the story and points a warning finger at those who countenance delay.

In the analysis of the thirteen deaths in our last series it is shown that an average of 16 hours and 10 minutes elapsed from the time of perforation to closure. If these patients could have been operated upon earlier it is needless to say many of them could have been saved.

Other factors that influence prognosis are the site and size of a perforation. The larger the hole the higher mortality. Gastric perforations have a higher mortality than duodenal because they tend to remain open; the mortality is higher in men than in women and the loss of tissue immunity is also a factor in higher mortality.

Treatment:—It is our hope that through the study of these three groups of cases, coming from one institution, operated upon by a few surgeons, that new light will be thrown on a subject that has been universally poor, and has caused concern

in the surgical world. Recently greater consternation has been thrown on this problem by a few isolated reports of spontaneous recovery in gastrointestinal perforation. That the conclusions of such reports are erroneous and ill-advised is eminently clear to anyone of major pathological or surgical experience. At this time we see no justification in discarding the practices or principles that we have followed for a quarter of a century. Experience proves that a ruptured ulcer, seen in the first 24 hours, can be treated with a lower mortality and morbidity by surgery. The treatment, therefore, is one of immediate surgical repair. Many of the authors, Moynihan, Allen and others had admonished against even the short delay in fluoroscopic examination. The diagnosis can be made readily in over 96 per cent of cases and the patient should be prepared immediately by:

(1) Gastric decompression—empty stomach and continue suction.
(2) Patient placed on his left side.
(3) Hypodermic of morphine and atropine.
(4) Start blood transfusion.
(5) Give 500,000 units of penicillin intravenously and repeat 200,000 units every four hours.
(6) Terramycin, 0.5 gram now and repeat every four hours.
(7) Intratracheal anesthesia, especially nitrous oxide and oxygen. We no longer prefer spinal anesthesia because of the shock state of the patient and the greater incidence of pulmonary complications.
(8) Right paramedian incision or transverse incision.
(9) Abdomen opened and pathology located.
(10) Tacking of a piece of living omentum over the perforation. Reinforce with interrupted plain silk sutures.
(11) Closure of peritoneum without drainage and closure of fascia with interrupted wire suture if perforation is less than 12 hours old. When it is over 12 hours old and a large number of bacteria are present the wire sutures are placed but not tied and a rubber tissue drain is put down to the peritoneum. This drain is removed and the sutures tied in 24 hours, (delayed closure). The spaces in the superficial fascia are obliterated and the skin closed with dermal suture.

Postoperative Treatment:—

(1) Levine tube with continuous Wangensteen suction.
(2) Nothing by mouth during period of acute ileus.
(3) Morphine for restlessness.
(4) Patient kept in water and electrolyte balance.
(5) Proper administration of glucose and sodium chloride.
(6) Blood transfusions—Maintaining a proper total protein level in the blood.
(7) Patient to sit on side of bed first day, out of bed second day.
(8) Fluids in small amounts when peristalsis returns.
(9) Intravenous administration of Vitamin C.
(10) Early ambulation (encourage early walking about). It is well to have on a supporting binder as a Scultetus when patient gets up.

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Discussion

Dr. Horace W. Soper (St. Louis, Mo.):—I should like to say one thing that will interest Dr. Goldstein, our Historian.

In 1917 William J. Mayo published a paper on the subject of lesions in the esophagus, stomach and duodenum. He had observed that the habit of taking hot foods and drinks was usually one of the things given in a history of gastric and duodenal lesions of these cases, whether they were perforations or not. He performed a large series of postmortems on mummies, subjects who lived prior to the discovery of fire, and found no lesions whatsoever in the esophagus, stomach or duodenum. He further found that Chinese men ate at the first table when the rice was hot. They had very frequent lesions in these regions; and the women waited for the second table and very rarely had them.

So that gastric and duodenal lesions, esophageal lesions, are largely diseases of civilization, since the discovery of fire.

I myself, in our histories of cases of this character, have found they usually gave a history of having eaten their food and drink hot.

Dr. Hyman I. Goldstein (Camden, N. J.):—Perhaps, the first recorded instance of surgical removal of the ulcer-bearing area of the stomach, appears on *"The Second Stéle of the Miraculous Cures Discovered at Epidaurus"*.

The man with an ulcer of the stomach is to be operated upon by the priests of the temple. "This man falls asleep and has a dream. It seems that the god orders the servants who accompany him to seize him and hold him firmly while he opens his abdomen. The man begins to flee, but the servants catch him and hold and bind him. But Aesculapius (i.e. the priest) opens his abdomen, accomplishes the excision of the ulcer, resews the abdomen, and delivers the man from his bonds. Immediately he goes out cured, and the stone pavement of the dormitory is covered with blood"! Footnote: This history is very remarkable, because it proves that the priests of Aesculapius, after having caused the patients to fall asleep, practiced real surgical operations on them and did not content themselves with impressing their imagination.

I need only refer to the early reported cases of perforated ulcer.

Dr. Frank R. Fabiani (Dobbs Ferry, N. Y.):—I should like to inquire as to the incidence of ulceration in the gastrointestinal tract following severe body burns.

Another question is the matter of continuous aspiration as a watchful method of treating a perforation. Has this procedure been sufficiently attempted and evaluated?

Dr. Libby Pulsifer (Rochester, N. Y.):—What I have to say is from a pretty puny experience as compared with the six hundred cases reported by the speaker, but I think we ought to provoke him a little bit.

We must guard against mistaking perforated ulcers for pancreatitis. In this day, house officers may quite frequently make blood amylase determinations on acute abdominal cases before the attending physician sees them. I think it should be emphasized that when presented with such a case, whose blood amylase has been found to be high, we should guard against being prejudiced in favor of the diagnosis of pancreatitis and so neglect to operate upon a possible perforated ulcer.

Dr. O'Donoghue said that in some of the cases, in which operation was delayed, he left drains. I wonder what comes out of the drain and whether he will keep up this practice.

Lastly, what is the sense of giving these people Vitamins B and C? They must have stored enough to last them the few days of their acute illness. It is done all around, I know, but is there any real justification for it?

Dr. A. X. Rossien (Kew Gardens, N. Y.):—I should like to know how frequently you encountered cases of acute perforation of peptic ulcers that are not operated upon and develop accessory pockets.

I have seen three cases; one on the lesser curvature of the stomach that had developed an accessory pocket by being walled off in the peritoneal reflections off the stomach that go into the making of the gastrohepatic ligament; one that perforated into the duodenal cap that was walled but resulted in marked upper gastrointestinal symptoms including severe vomiting for two years; the third case of perforated ulcer of the duodenal cap apparently was not completely walled off at first and as a result a sinus tract going diagonally and downward into the left pelvis was found at operation.

Dr. John B. O'Donoghue (Chicago, Ill.):—In answer to the doctor's question relative to the occurrence of gastrointestinal ulceration in burned patients, I wish to say that the literature is full of examples of the occurrence of these ulcers, and Dr. Ivy, in his new book on peptic ulceration, has called attention to the frequency of multiple erosions. He pointed out they frequently go only through the mucosa and submucosa. The superficial erosions of the mucosa and the deeper ulcers are one and the same lesion, differing only in degree, depending on the variation of the tissue's susceptibility, angiospastic factor, amount of histamine or histamine-like substance present, the presence of varying amounts of depressor substance, or the amount and concentration of acid. The histamine liberated by the destroyed protein has been shown to cause the renal complications in burns.

The second question: "Can one ever aspirate these patients, and then with continuous suction carry them along under observation for awhile?" In a large number of cases this will seriously increase the mortality and mislead the doctor by some slight temporary improvement; creating the feeling that the ulcer had sealed off, hence lose the all-important safeguard, namely, early operation, especially in the first six hours. No one can predict the size of the ulcer, the amount of peritoneal soilage, or the reaction of the tissue to chemical or bacteriological contamination, therefore the only safe, logical treatment must be to see the perforation and close it. We can never predict which ulcer may be sealed off.

Dr. Goldstein's early reference to Rudolph Valentino is an example of one that we wish to God had been sealed off, but instead he was operated on and there was found that characteristic dishwater fluid around his appendix, its tip reddened by the irritating fluid in which it was floating. The surgeon was misled by his findings and did not realize that instead of having appendicitis he had a ruptured duodenal ulcer with spillage along the right colonic gutter. As I said to you, if you

will first take our three series, and if you will review the literature, you will see that when these people are operated on early the mortality is lower.

The doctor mentioned acute pancreatitis, and I think that we all will agree that acute pancreatitis is certainly increasing in incidence, at least it is being diagnosed more frequently. We are reminded, however, that it occurs more frequently in women who have had some previous digestive history which might refer itself to the gallbladder tract and which has an early rise in the blood amylase. I think you saw it in the third place on my slide on differential diagnosis.

In answer to the question on the average hospital laboratory not doing the amylase test: This is a simple procedure that is easily learned and can be completed in thirty minutes while the patient is being prepared for surgery. So that this may be done at night, the surgical residents are prepared to perform the test.

To the further question about the incidence of acute pancreatitis, I believe from our material at least that acute appendicitis and coronary thrombosis occur more frequently.

The question of drainage of the peritoneum has been a controversial subject for a great many years. I agree with those in medicine who believe that the peritoneal cavity cannot be drained, and therefore close the peritoneum without drainage. In cases that are operated near twelve hours or later when there is bacterial contamination, one might consider draining the abdominal wall or delayed closure of the abdominal wall. However, with antibiotic therapy, abdominal wall abscess and infections are rare. In grossly infected cases drainage of the abdominal wall for forty-eight hours should be considered.

To the question about use of vitamins postoperatively I would like to say that Vitamins B and C are used in large doses as we know they play an important part in wound healing. By a grant from Hoffmann-LaRoche, working through the Hektoen Institute, I studied the presence of Vitamin C in ulcer cases and was able to prove that in every case of carcinoma of the upper gastrointestinal tract there was no Vitamin C in the blood. I was able to show an absence or a deficiency of Vitamin C in all cases of gastric and duodenal ulcer. We showed that in carcinoma, no matter how much Vitamin C was given by mouth, the level of Vitamin C was never raised. However, it was raised when it was given by hypo and one could bring it up to the normal level.

I believe that the case as cited by my colleague is one of a long standing forme fruste ulcer with perforation and a multilocular cavity formed with communicating sinus to the outside.

In conclusion I wish to thank the many participants in the discussion for their pertinent comment and finally, in closing I wish to publicly express my respect and appreciation to my associate of fourteen years, Dr. Maurice B. Jacobs, who died suddenly while working on this paper.

THE EFFECT OF ANTACID PREPARATIONS ON SERUM AUREOMYCIN LEVELS

MORTIMER LEE WILLIAMS, M.D.*
and
DON JAMES WEEKES, M.D.*
Baltimore, Md.

The therapeutic use of aureomycin† frequently causes troublesome gastric irritation. Recently several investigators have reported that the antacid preparations used to prevent or treat gastric irritation interfere with the absorption of aureomycin[1,2]. Before we were aware of these reports, we had treated with an antacid 20 patients who had nausea or vomiting, usually on the second or third day following the administration of aureomycin. The results in these cases had been uniformly good, both for the primary disease and the side-effects.

TABLE I

Subject	Sex	Blood levels of aureomycin in Micrograms per milliliter of serum	
		with Mucotin	without Mucotin
S. W.	f	1.0	0.5
W. L.	m	1.0	1.0
D. B.	m	0.5	0.5
M. M.	f	0.5	0.5
L. W.	m	0.5	0.5
F. L.	m	1.0	0.5
D. W.	m	1.0	1.0
L. H.	m	1.0	1.0
M. P.	f	1.0	1.0
M. S.	f	0.5	1.0
M. T.	f	0.5	not done
N. F.	m	1.0	1.0
J. L.	f	1.0	1.0
F. C.	m	1.0	1.0
M. C.	f	0.5	not done

In order to rule out the possibility that aureomycin had controlled the symptoms of the disease prior to our administration of the antacid, it became necessary to determine blood levels of aureomycin, both with and without the administration of

*Johns Hopkins Hospital, Department of Otolaryngology.
†The aureomycin was supplied by Lederle Laboratories Division, American Cyanamid Company.

antacid preparations. For the purpose of this investigation we used Mucotin®**, a brand of gastric mucin antacid mixture and a brand of aluminum gel that is also antacid. The method of bacterial assay described by Dornbush and Pelcak[3] was used to determine serum levels. Under fasting conditions aureomycin was given without an antacid drug in one group; in a second series Mucotin was given with aureomycin and in a third series aluminum gel was given with aureomycin.

A tabulation of serum levels of aureomycin with and without Mucotin (Table I) shows the following: out of 15 patients, the blood level in 10 was the same with Mucotin or without it; in two cases a higher blood level was obtained using Mucotin, and in one case the blood level was lower; two cases were unavailable for comparison. Two tablets of the antacid preparation were chewed thoroughly with-

TABLE II

Subject	Sex	Blood levels of aureomycin in Micrograms per milliliter of serum	
		Aluminum gel and aureomycin Simultaneously	Aluminum gel one half hour before
M. P.	f	less than 0.06	
J. L.	f	less than 0.06	
D. B.	m	less than 0.06	
W. L.	m	less than 0.12	
M. S.	f	0.5	
L. H.	m	0.25	
M. M.	f		0.5
N. F.	m		0.5
D. W.	m		0.5
F. C.	m		0.5

out water, and then nothing was given by mouth until the aureomycin was administered thirty minutes later.

A brand of aluminum gel was used in the third series. Four members of the group took one ounce of this preparation 30 minutes before taking the aureomycin, and six members took simultaneous doses of aureomycin and aluminum gel. Table II shows that the four patients taking aluminum gel 30 minutes before aureomycin all had a blood level equal to the lowest in the Mucotin table; in the group of six people taking aluminum gel simultaneously with aureomycin, five had a blood level considerably lower than that where aureomycin was used alone or with Mucotin, while one patient equalled the lowest blood level when Mucotin was administered.

As far as practical the same healthy adults were used for each series and at least one week elapsed between each series, during which time the patient received

**This study was aided by a grant from The Harrower Laboratory, Inc., who also supplied the Mucotin.

no aureomycin. The routine procedure was to give 11 milligrams of aureomycin in one dose per kilo of body weight at 9:30 A.M. and five hours later to take blood for analysis.

We feel that these results are significant even though the series is small. The blood levels of the patients taking Mucotin and aureomycin were identical with the blood levels of the patients who received aureomycin alone. Strangely enough the blood levels were higher in two cases when Mucotin was used than when aureomycin was given alone, possibly due to the decreased gastric irritation, lowered gastric motility and increased absorption of aureomycin. Our results when aluminum gel and aureomycin were administered simultaneously or within a few minutes of each other agree with those of Seed and Wilson[1], namely that the antacid interferes with the absorption of aureomycin. In only one case was a high serum concentration of aureomycin obtained, and then only 0.5 micrograms per milliliter of serum. When there was a delay of 30 minutes, however, between the administration of aluminum gel and aureomycin an average or maximum serum level was always obtained. When aluminum gel was used in this way the levels were uniformly higher, but in no case was a level of 1.0 micrograms obtained, which was the maximum level when Mucotin was used.

We conclude from this study that the delay of 30 minutes between the administration of the antacid and aureomycin is a factor in obtaining higher blood levels, and further that the antacid preparation (Mucotin) does not lower, but on the contrary, seems actually to increase the aureomycin blood level in some cases. At the same time Mucotin relieves the annoying side-effects of nausea and vomiting, which sometimes accompany aureomycin administration.

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INTESTINAL SYMPTOMS IN NONGASTROINTESTINAL LESIONS

BERNARD J. FICARRA, M.D.*
Brooklyn, N. Y.

Many pathologic conditions masquerade under symptoms which are not directly associated with the organ involved. The gastrointestinal tract is the frequent victim of symptoms which are not indicative of digestive disturbances. The abnormal stomach expresses itself most often with the symptoms of nausea and vomiting. If a patient presents the symptoms of nausea and/or vomiting attention is immediately attracted to the gastrointestinal tract. However, thought must also be given to those pathologic entities not directly associated with the gastrointestinal tract, but which often express themselves with gastrointestinal complaints.

Among the frequent causes of extragastrointestinal nausea and/or vomiting are the following (according to regions).

1. Head and Neck:—

1. Fracture of skull
2. Cerebral concussion
3. The meningitides
4. Cerebral vascular accidents
5. Sinus thrombosis
6. Cerebral tumor and abscess
7. Ménière's Disease
8. Hydrocephalus
9. Epilepsy
10. Cerebral syphilis
11. Migraine

Acute vomiting with or without nausea in cerebral lesions occurs suddenly. In trauma to the skull vomiting may occur during the stage of cerebral irritation. Vomiting is rarely a solitary symptom in the above pathologic entities.

Associated symptoms are headache, vertigo, restlessness, delirium convulsions, eye changes, etc.

2. Thoracic Lesions:—

1. Cardiac pathology
    a. Congestive heart failure
    b. Acute toxic myocarditis
    c. Coronary thrombosis
2. Mediastinal tumor
3. Intercostal neuralgia
4. Lung diseases (tuberculosis, cancer, bronchiectasis, gangrene)

Diseases of the heart frequently produce nausea. Especially is this true in congestive heart failure when visceral passive congestion disturbs digestion. Coronary thrombosis is often associated with vomiting. The frequency of death due to "acute

*Department of Surgery, St. Peter's Hospital, Brooklyn, N. Y.

indigestion" is recorded in the daily press. In these instances the real cause of death is coronary occlusion. Pulmonary and mediastinal pathology produce gastric symptoms. The method whereby this occurs is not exactly known. However, it may be due to pressure on the vagus nerve.

3. Abdominal Pathology:—

1. Retroperitoneal Hemorrhage
2. Injuries to, and diseases of the spleen
    a. Rupture of spleen
    b. Splenic infarction
    c. Banti's disease
3. Hematomyelia
4. Tubo-ovarian and uterine disease
    a. Cyst (rupture or twisted pedicle)
    b. Hemorrhage
    c. Salpingitis
    d. Pelvic peritonitis
    e. Ectopic pregnancy
5. Diseases of urinary bladder
6. Subphrenic abscess
7. Aneurism of abdominal aorta

Certain injuries which produce hemorrhage result in symptoms of nausea and vomiting. Intraabdominal hemorrhage irritates the gastrointestinal tract thus producing gastrointestinal symptoms. Examples of this type are hemorrhages resulting from rupture of the spleen, twisted uterine fibroid or ovarian bleeding. Other types of hemorrhage such as retroperitoneal and bleeding in and about the spinal cord, produce nausea and vomiting by irritating spinal nerves.

When the spleen enlarges greatly due to Banti's disease, leukemia, etc., it may cause nausea and vomiting by direct pressure on the gastrointestinal tract or by venous congestion.

Localized inflammatory processes may cause nausea or vomiting via peritoneal irritation. Examples of this type are subphrenic abscess and pelvic peritonitis as from salpingitis or severe cystitis.

4. Renal Pathology:—

1. Nephrolithiasis
2. Acute and chronic glomerulonephritis
3. Congenital anomalies
4. Polycystic Kidneys
5. Hydronephrosis
6. Renal disturbances during pregnancy

Medical diseases of the kidneys are commonly associated with nausea and vomiting. The same is true with congenital anomalies, polycystic kidneys and renal calculi. Many a surgeon has been embarrassed by performing a laparotomy for an acute condition within the abdomen only to ascertain later that severe renal pathology was the cause of the presenting symptoms.

Renal conditions associated with the toxemias of pregnancy are a common producing cause of vomiting. Nausea itself or nausea with vomiting are common in the early months of normal pregnancy. When the simple nausea of pregnancy becomes severe it may be termed, "pernicious vomiting of pregnancy". This type of vomiting may persist for weeks.

5. Injudicious use of drugs:—

1. Emetics (apomorphine, ipecac, antimony)
2. Morphine addiction
3. Tobacco
4. Acute alcoholism
5. Digitalis Intolerance
6. Barbiturate poisoning

Certain drugs as apomorphine are employed to produce vomiting. Other drugs such as morphine may cause vomiting as a toxic manifestation. One of the withdrawal symptoms in morphine addicts is vomiting. The use of tobacco in persons not accustomed to smoking may cause vomiting. The same is true of nonalcoholics who overindulge in drinking. An overdose of certain drugs such as digitalis, phenobarbital, ergot, etc., may produce vomiting.

6. Diseases of the blood:—

1. Acute secondary anemia
2. Pernicious anemia
3. Henoch's purpura
4. Sickle-cell anemia

Disturbances in the hematopoietic system may cause gastrointestinal symptoms as seen in the "crises" of sickle-cell anemia. Other dyscrasias such as pernicious anemia are associated with nausea and vomiting. However these symptoms may be attributed to achylia gastrica which accompanies the anemia. In Henoch's purpura, nausea is often associated with abdominal symptoms closely paralleling intussusception.

7. Metabolic Diseases:—

1. Exophthalmic goiter
2. Diabetes mellitus
3. Addison's disease
4. Angioneurotic edema

Some disturbances in endocrinology and metabolism result in vomiting. In diabetics these symptoms are characteristic of acidosis as it becomes more prominent. In exophthalmic goiter the gastric mucosa is irritable and frequent episodes of vomiting accompany exacerbations of the disease. In Addison's disease the gastric symptoms are spontaneous and variable throughout the course of the disease. In allergic manifestations, as angioneurotic edema, the external swelling may be associated with a similar reaction internally and will produce vomiting, so-called intestinal allergy.

8. Neuropsychiatric Disturbances:—

1. Tabes dorsalis
2. Epilepsy
3. Dysmenorrhea
4. Neurosis
5. Severe psychic shock or fright
6. Seasickness and airsickness

Neurological disorders are frequently associated with nausea, vomiting and abdominal pain. The gastric crisis of tabes dorsalis is well known. Occasionally epileptic attacks are associated with vomiting especially when the attack occurs soon after eating. Dysmenorrhea may be so severe that nausea and vomiting may accompany the first menstrual days.

Psychosomatic disturbances are notorious for the production of gastric symptoms. The neurotic type of vomiting may occur repeatedly day after day, sometimes after each meal, sometimes only once a day. Severe fright may produce vomiting as a reactionary symptom. The nausea and vomiting of seasickness are too well known to necessitate further discussion.

Summary and Conclusions

1. Thought is given to the cardinal gastrointestinal symptoms of nausea and vomiting.

2. A listing of nongastrointestinal conditions producing these symptoms is given.

3. This presentation emphasizes many lesions which mimic gastroenterologic disturbances, thereby camouflaging a true diagnosis.

4. This topic emphasizes the importance of the gastrointestinal tract as the means of expression in both organic and psychosomatic diseases.

5. Recalling to mind these extragastrointestinal diseases will often assist the gastroenterologist in arriving at an accurate diagnosis more easily.

ESOPHAGEAL HIATUS HERNIA

Report of a Case With Multiple Associated Abnormal Findings

EMANUEL W. LIPSCHUTZ, M.D., F.A.C.P.
Brooklyn, N. Y.

Esophageal hiatus hernia, either with or without associated pathology in the duodenum or gallbladder, is encountered not infrequently in a routine x-ray survey of the gastrointestinal tract. The association of the multiple findings in the case to be reported here, however, it not too common and is interesting enough to warrant a report.

Mrs. J. G., age 71, was first seen April 30, 1949, complaining that for the past year and a half, she has been experiencing attacks of epigastric pressure and distress which usually come on after supper and especially when she assumes a reclining position. At times, the epigastric distress awakens her from sleep forcing her to walk about for hours before she is relieved. In the past two weeks she experienced two such attacks associated with nausea and requiring an opiate for relief. Associated symptoms are regurgitation of food, belching after meals, anorexia and loss of seven pounds in the past few weeks. She also noted a tendency to constipation.

Past History:—About sixteen years ago she had a growth removed from her tongue and twelve years ago she was told she had gallstones.

On physical examination the heart and lungs were essentially normal. There was no lymphadenopathy and no peripheral edema. The abdomen was soft and the liver edge was palpable about two finger breadths below the costal margin in the right midclavicular line. A poorly defined, rather hard and somewhat tender mass moving with respiration, was felt in the right upper abdomen below the liver edge. It was difficult to determine whether it was due to an enlarged gallbladder or a right kidney. Rectal examination was negative.

Laboratory findings:—Urinalysis essentially negative. Hemoglobin—90 per cent (16 gm.); red blood count—4,020,000; leucocytes—5,600. Segmented—60 per cent; lymphocytes—40 per cent. Erythrocyte sedimentation rate—12 mm. per hour (Westergren). Gastric analysis—Amount extracted was insufficient for quantitative determinations; free HCl was present; blood absent. Blood chemistry revealed glucose—102 mg. per 100 c.c. blood (fasting), Urea N—18.6 mg.; calcium—9.4 mg. X-ray examination of the gastrointestinal tract including a cholecystographic study (Figs. 1A, 1B and 1C) revealed an extensive hiatus hernia with about one-half of the stomach lying within the thoracic cavity; a shortened esophagus is suggested from the film as a possible contributory factor in the gastric herniation. The x-rays also reveal a duodenal ulcer, cholelithiasis (Fig. 1B) and a large stag-horn calculus in the right kidney.

Comment:—As mentioned above, esophageal hiatus hernia is not infrequently associated with gallstones or peptic ulcer or even colonic diverticulosis. According to von Bergmann[1] disease of the gallbladder or any other part of the gastrointestinal tract may act as a source of irritation sufficient to initiate repeated reflex contractions of the longitudinal muscle fibres of the esophagus, thereby intermit-

tently pulling up the stomach through the esophageal hiatus and causing a permanent herniation, if the condition prevails long enough. It is also very likely that a congenital weakness of the diaghragm, such as an abnormally wide hiatal opening, may act as a contributing factor, although the condition may not become manifest until late in life. In some cases, although rarely, a congenital short esophagus may cause the stomach to occupy a position in the lower thorax. According to Held and Goldbloom[2] a congenitally short esophagus as such, does not exist. They offer the interesting hypothesis that a hiatus hernia is the result of primarily an enlarged esophageal hiatus which induces gastric herniation and that contraction and shortening of the esophagus is secondarily produced by vagus irritation in the hernia. It is generally agreed that circumstances which increase markedly intraabdominal pressure, such as constipation, chronic cough, repeated pregnancies, etc. may be important factors in the production of hiatus hernia in individuals predisposed to it.

Fig. 1A Fig. 1B Fig. 1C

In the case herein reported one is very much tempted to invoke the theory proposed by von Bergmann. The patient obviously has ample associated pathology in the duodenum, the biliary tract and even in the right urinary tract, to have induced reflex irritation of the esophagus over a period of years, resulting in a permanent hiatus hernia. As to the association of cholelithiasis with the marked calcification of the right kidney, one may only conjecture that the patient at some time in the past may have been suffering from a hyperparathyroidism or a parathyroid adenoma with a hypercalcemia and the resultant pathology. There is nothing at present in the clinical or laboratory findings to suggest parathyroid hyperfunction.

Summary:—A case of an esophageal hiatus hernia complicated by a duodenal ulcer, cholelithiasis and a large stag-horn calculus in the right kidney is here reported. The various theories as to the factors which may be operative in the production of hiatus hernia are described. The possibility of a complicating hyperparathyroidism in the past and the reason for such possibility is given.

REFERENCES

1. von Bergmann, G.: Functionelle Pathologie. Berlin, J. Springer, 1932, p. 68.
2. Held, I. W. and Goldbloom, A. A.: Hiatus Hernia. Rev. Gastroenterol. 3:291, (Dec.), 1936.

CHAPTER ACTIVITIES

New Jersey Chapter

A meeting of the New Jersey Chapter of the National Gastroenterological Association was held at the Academy of Medicine in Newark, on 27 November 1950.

The chapter voted to put on a group exhibit at the New Jersey State Medical Society meeting and appointed a committee consisting of Drs. S. William Kalb, Chairman; Louis L. Perkel, Hyman I. Goldstein, Leo H. Siegel, Harrison R. Wesson and Andrew J. V. Klein for this purpose.

Dr. Alexander Strelinger presented an interesting case of "Internal Hemorrhage Due to Diffuse Plasmocytoma".

At the meeting held at the Jersey City Medical Center on 22 January 1951, the program consisted of a symposium on "Unusual Gastrointestinal Diagnostic and Surgical Problems".

New York Chapter

The New York Chapter of the National Gastroenterological Association met at the New York Academy of Medicine on 8 January 1951.

Dr. Arthur J. Bendick spoke on "Advancement in the Roentgenological Study of the Duodenum"; Dr. Charles A. Flood spoke on "Benign Diseases of the Gastric Antrum" and Dr. Scudder Winslow presented: "Surgery in the Acute Phase of Ulcerative Colitis".

The next meeting of the Chapter will take place at the New York Academy of Medicine on Monday evening, 12 March 1951. A symposium on Carcinoma will be presented by members of the staff of the Memorial Center for Cancer and the Pack Medical Group.

Members of the medical profession are cordially invited to attend.

On Monday, 9 April 1951, the Chapter will hold its second annual clinical session. This session will be open to the members in good standing of the New York Chapter only and will feature interesting case presentations.

Those interested in participating should communicate immediately with the Chairman of the Chapter Program Committee, Dr. Franz J. Lust.

Philadelphia Chapter

A meeting of the Philadelphia Chapter of the National Gastroenterological Association was held at the Hahnemann Medical College in Philadelphia on Wednesday evening, 18 January 1951. The guest speaker was Dr. Charles W. Mayo of the Mayo Clinic, Rochester, Minn.

Wilkes-Barre-Scranton Chapter

The Wilkes-Barre-Scranton Chapter held a meeting at the Fox Hill Country Club in West Pittston, Pa. on 17 December 1950.

Dr. Edmund Matys of Dupont, Pa., was elected president to succeed Dr. Samuel Friedmann of Scranton, Pa. and Dr. E. Merton Hill of West Pittston, Pa. was reelected secretary-treasurer.

NEWS NOTES

Meeting of the Executive Committee

A meeting of the Executive Committee of the National Gastroenterological Association was held in New York City on Sunday, 5 November 1951.

Routine administrative matters were discussed and disposed of. Dr. Roy Upham, Secretary-General, reported upon the status of the Chapters and their activities.

The committee on office space rental reported that new and larger quarters had been obtained for the headquarters office. The new space is in the same building, on another floor.

The matter of considering applications for membership and advancement semi-annually was discussed, and it was unanimously agreed to follow this procedure in the future.

The Committee voted to increase membership, associate fellowship, fellowship, initial, and advancement fees, effective 1 January 1951, because of the rising costs of rents and services.

Dr. C. J. Tidmarsh, president, presented the following suggestions for the standing committees, which suggestions were approved by the Executive Committee.

Program Committee:—Dr. C. J. Tidmarsh, Montreal, Canada, chairman; Dr. William W. Lermann, Pittsburgh, Pa.; Dr. Sigurd W. Johnsen, Passaic, N. J.

Membership Committee:—Dr. F. H. Voss, Phoenicia, N. Y., chairman; Dr. H. E. De Walt, Pittsburgh, Pa.; Dr. Lynn A. Ferguson, Grand Rapids, Mich.; Dr. Arthur Kirchner, Los Angeles, Calif.

Editorial & Publication:—Dr. Samuel Weiss, New York, N. Y., chairman and Editor of The Review of Gastroenterology.

Endowment Fund Committee:—Dr. William C. Jacobson, New York, N. Y., chairman.

Convention Arrangements Committee:—Dr. Samuel Weiss, New York, N. Y., chairman; Dr. Sigurd W. Johnsen, Passaic, N. J.

Research Committee:—Dr. Henry A. Rafsky, New York, N. Y., chairman.

Other committee appointments were left for a future meeting.

Because of a vacancy occurring in the membership of the Council, under the provisions of the Constitution and By-Laws, Dr. Lynn A. Ferguson, of Grand Rapids, Mich. was elected by the Committee.

The Boston, Argentina, New Jersey and New York Chapters presented for ratification the applications of the following, which applications were approved by them and accepted by the Executive Committee as indicated: Dr. Ernesto Brik,

Mar del Plata, Argentina, Member; Dr. Frank M. McCarthy, Lawrence, Mass., Associate Fellow; Dr. Louis A. Perrotta, Bronx, N. Y., Member; Dr. Rafael A. Jacobo, New York, N. Y., Member; Dr. Harold B. McCluskey, East Orange, N. J., Member.

The following were elected to membership-at-large in the National Gastroenterological Association: Dr. Saul Fortunoff, Butler, Pa., Member; Dr. Gustav Zechel, Chicago, Ill., Fellow; Dr. Edward P. Thomas, Indianapolis, Ind., Member; Dr. Roland Gagne, Ottawa, Canada, Member, and Dr. Albert A. Sichel, Larchmont, N. Y., Member.

Upon presentation of the necessary qualifications, the following were advanced as indicated: Dr. Sidney Winters, New Haven, Conn., Associate Fellow; Dr. F. R. De Vincenzo, Hoboken, N. J., Associate Fellow.

Teaching Seminar in Proctology

The International Academy of Proctology will present its first teaching seminar on proctologic subjects, including the more recent developments, in the form of a symposium and round-table discussion.

The session will be held in New York City, April 7, 1951.

Registration for the seminar will be open to licensed physicians who are members of the American Medical Association; State or County Medical Associations and graduates of an approved medical school. Admission to the seminar will be by card only.

For registration or further informaton communicate with Dr. William Lieberman, Chairman, Seminar Committee, International Academy of Proctology, 1819 Broadway, New York 23, N. Y.

Correction

In the article "Results of Peptic Ulcer Treatment with Protein Supplements" by Dr. Lester M. Morrison, which appeared in the November 1950 issue of The Review of Gastroenterology, the title of Table I, which is on page 1059 should be: Description of Clinical Material and Results of Peptic Ulcer Therapy Using Whole Protein Supplement.

In the same Table, under Case 3, the comment should be: "Placed on bland diet with mid-meal protein supplement feedings—3 months".

In the second line on page 1065, the word *hydrolysate* should read: *protein supplement.*

ABSTRACTS

GASTROINTESTINAL TRACT

THE USE OF CO_2 AND AIR AS ANTISPASMODICS. Paul Cane. Brit. J. Radiol. 22:216-223, (April), 1949.

The technic of relaxing spasm of the gastrointestinal tract by means of inflation with air or carbon dioxide is described. Seidlitz powder can be used.

For relaxation of spasm in the esophagus enough fluid barium is given to locate the site of the narrowing. The patient is then given a small amount of Seidlitz powder in a small quantity of water, followed by a second dose of Seidlitz powder. Screening should follow shortly after the ingestion of the powder as the resulting relaxation is very often rapid. This technic is useful in achalasia of the cardia and also in organic strictures. In these cases, where the passage of barium is insufficient to visualize the extent of the lesion, the distention caused by the gas will produce a degree of relaxation, permitting complete visualization.

This technic is also useful in the stomach and colon. For this purpose it is advisable to mix the powder with the barium suspension.

If the antrum is to be inflated the patient is to be examined in the left oblique supine position.

Pyloric and prepyloric spasm will be relaxed, allowing clear visualization of any existing lesion. This technic will produce a double-contrast effect, by means of which doubtful outlines of the greater and lesser curvatures may be easily studied.

Seventeen radiograms are published. ADOLPH ABRAHAM

CONGENITAL DIAPHRAGMATIC HERNIA ON THE RIGHT SIDE. J. M. Boyles. Northwest Med. 48:324-326, (May), 1949.

Congenital diaphragmatic herniae are rare. Most of them pass through the pleuroperitoneal canal and very few have a hernial sac. In their presence any of the abdominal organs may be found in the chest. In a series studied in the University of Minnesota about one in one hundred thirty-nine autopsies on infants up to one year of age revealed diaphragmatic hernia.

Due to gas distention in the herniated viscera dyspnea and cyanosis develop. Tachycardia, regurgitation and vomiting of bile stained material develop in practically all cases.

The author gives a case history of a new born female illustrating the symptomatology and the x-ray findings in his case; the hernia was on the right side of the diaphragm. No diaphragm was found covering the posterior right half so that at operation the anterior diaphragmatic flap was drawn posteriorly and sutured. Complete recovery of the infant is reported. A. X. ROSSIEN

ROENTGEN STUDIES OF THE UPPER GASTROINTESTINAL TRACT IN VAGOTOMY. Frank Isaac, Richard E. Ottoman and Joseph A. Weinberg. Am. J. Roentgenol. 63:66, (Jan.), 1950.

Eighty-three consecutive cases of vagus resection were studied roentgenologically. The post-vagotomy studies included 75 patients at one week, 72 patients at one month, and 50 patients at 6 months. The changes found in the stomach after vagus resection are decreased peristalsis, delayed motility, and loss of tone with dilatation. Of those examined one week after vagotomy, 87 per cent showed decreased peristalsis, 80 per cent delayed motility, and 54 per cent loss of tone. The tendency for these functions is toward gradual improvement. At 6 months, however, a rather large percentage did not return to normal: 48 per cent in case of peristalsis, 41 per cent in case of motility, and 33 per cent in case of tone. The changes are found with equal frequency whether the operation has been performed by the thoracic or abdominal route and whether it is simple vagotomy or combined with gastroenterostomy. The tendency to return to normal, however, is greater after the combined operation than after simple vagotomy. A majority of those having demonstrable ulcer crater in the duodenal bulb before operation show a disappearance of the niche after vagotomy. In this series 42 per cent had a crater before operation, but only 14 per cent after vagotomy.

In the small intestine the most constant changes consist of dilatation of the duodenum and delay of the intestinal motility. Less constant alterations of small intestinal pattern include dilatation of the jejunum and ileum, segmentation, pooling and flocculation of the barium. FRANZ J. LUST

STOMACH

CARCINOMA IN A HERNIATED GASTRIC CARDIA ASSOCIATED WITH SHORT ESOPHAGUS. J. Dawson. Brit. J. Radiol. 23:270-273, (May), 1950.

A rare case of adenocarcinoma in a herniated gastric cardia associated with short esophagus is described. The diagnosis was made by radiological examination, esophagoscopy and histological biopsy. The etiology is discussed.

Cancer formation in this case may be the result of cardiac gastritis. The literature is reviewed.

Two roentgenograms and two photomicrographs are published. ADOLPH ABRAHAM

CYTODIAGNOSIS IN GASTRIC CARCINOMA. Lois A. Platt. Postgrad. Med. 7:26-32, (Jan.), 1950.

The author reviews the difficulties in preparing and interpreting gastric washings in the detection of gastric cancer. A detailed method of preparation of the smear is given and five cases are reported in which three well-developed cancers were definitely corroborated by the smear examination. No early cancers of the stomach have been detected by this method. The author advocates a possible screening test of this sort in men over the age of thirty and women after the age of fifty-five, as well as in those cases of low gastric acidity. The author emphasizes the importance of proper preparation of the smear and the amount of time consumed in proper interpretation of a satisfactory smear, and concludes that it is a useful aid in diagnosis of cancer of the stomach, but it is not infallible and that the methods of preparation and examination of the smear are time consuming, consequently expensive and cannot therefore be used as a screening test or as a routine test in cancer detection at the present time. JACOB A. RIESE

DUODENUM

THE MANAGEMENT OF BLEEDING DUODENAL ULCERS. Robert W. Fraser and John P. West. Ann. Surg. 129:299, (March), 1949.

The total mortality in a group of 177 consecutive patients suffering moderate to severe hemorrhage, from duodenal ulcer, was 6.2 per cent. The nonoperative treatment of 165 patients resulted in 7 deaths, a mortality of 4.2 per cent. Operative treatment of 12 patients suffering from severe and uncontrolled hemorrhage was followed by 4 deaths, a mortality of 33.3 per cent. The operation apparently saved the lives of four patients in this series but at the same time it appears probable that some of the patients subjected to operation might have survived without surgical intervention. The occasional indications for surgical treatment of bleeding duodenal ulcer are limited with few exceptions to patients over 50 years of age. FRANZ J. LUST

INTESTINES

TORSION OF THE APPENDICES EPIPLOICAE. Philip Ladin. N. Y. State J. Med. 49:2168, (Sept. 15), 1949.

The torsion of an appendix epiploica as a cause of acute abdominal signs and symptoms is not usually considered. Inasmuch as five cases were found in the records of a small hospital, this condition may be more common and should be looked for more often. In a patient who presents a long history (two or more days) and whose physical findings are relatively mild this condition should be suspected. If the clinical condition warrants surgical intervention, it should be looked for at operation. Removal of the appendix epiploica should be done with careful dissection of the base. The literature was reviewed briefly, and five cases were reported in this paper bringing the total number of reported cases up to 73. FRANZ J. LUST

"ELEOMA" OF THE RECTUM. William B. Swigert. Rocky Mountain M. J. 46:739-740, (Sept.), 1949.

Tumors following therapeutic rectal injection of oils, chemicals, or other substances are not uncommon.

Swigert cites a case of his own which was diagnosed originally, after biopsy, as lymphangioma: but following a proctectomy with full dissection, the pathology was found to be an "eleoma of the rectum".

While the immediate results are good, the author fears they may be only temporary. The proctectomy was performed in the erroneous belief that the case was one of lymphangioma, and Swigert feels that such surgery is only justified if conservative measures prove to be unavailing. A thorough trial should be given such measures as hot saline irrigations through the rectum and vagina.

He agreed with R. I. Jackson that the use of oil base sclerosing agents in the injection treatment of hemorrhoids should be condemned. REGINALD B. WEILER

SURGERY OF ILEAL TUMORS—REPORT OF A CASE OF INTUSSUSCEPTION LED BY LIPOMA OF ILEUM. Roland L. Grausman and Seymour S. Rogers. Am. J. Surg. 77:387, (March), 1949.

The terminal ileum is the most common site of tumors of the small intestine. Most of these tumors are benign and their clinical importance depends upon the complications of obstruction, necrosis or intussusception. These tumors are diagnosed by symptoms of vague, colicky, intermittent, pain in the right lower abdominal quadrant. The pain increases in intensity with the onset of the aforementioned complications. Roentgenograms and particularly fluoroscopy are useful aids in diagnosis. The suggestive radiographic signs are filling defects, deformity of bowel outline and evidence of distended bowel. The treatment is surgical and consists of enterostomy and exclusion, reduction of an intussusception and enterostomy, or resection of the tumor-bearing area.

A case of irreducible, ileocolic intussusception caused by a lipoma of the ileum and treated by resection of the affected bowel and an ileotransversostomy, is described. FRANZ J. LUST

THE DELAYED TREATMENT OF APPENDICEAL ABSCESS. Philip Ladin. New York State J. Med. 50:681-685, 1950.

The delayed treatment of peritonitis of appendiceal origin dates back to 1901 when Ochsner first described it. At that time he reported striking improvement over the usual results of his day. There have been scattered reports since then of successful treatment by this method. Recent experimental work has shown that high concentrations of penicillin in the blood and serous cavities inhibits the growth of B. coli. It has also been shown that the concentration in ascitic fluids and blood is directly proportional to the dosage of penicillin given intramuscularly and intravenously. The theory was postulated that B. coli inactivates penicillin and therefore massive doses are necessary to overcome this inhibitory effect. It seemed logical that combining Ochsner's method with present day supportive treatment and antibiotics should give the best results. The peritoneum is well able to handle the infection if not interfered with, and these patients can then be operated upon at a future date with minimal risk. Three of the patients reported showed an appendix divided into two halves with the ends sealed off and no evidence of leakage. The author points out that the readiness with which these patients return for further surgery has been noted by others as well as by him. Four to six weeks appears to be a safe time for the secondary operation under this regime of treatment and primary union can be obtained in these patients. The only disadvantage to this form of treatment is that it requires careful and frequent observation, and the exercise of good clinical judgment, as to when as well as, whether or not to intervene. Six cases of appendiceal abscess treated by this method are reported in detail. The patients varied in age from two to forty-six, and presented a variety of clinical pictures. The temptation to interfere surgically is probably so great that it is a major factor in the failure of this method of treatment, to become more popular in spite of repeated reports as to its superiority.

FRANZ J. LUST

RECURRENT SMALL INTESTINAL INTUSSUSCEPTION IN CHILDREN. M. D. Teitelbaum and Nathan Arenson. Am. J. Roentgenol. 63:80, (Jan.), 1950.

Five cases of small intestinal intussusception are described. This condition in young children is more frequent than is commonly believed. It is presumably a manifestation of neuromuscular dysfunction and, in some instances, may be an allergic response. The intussusception, which is transient, recurrent and usually involves multiple segments of the small intestine, produces rather constantly, intermittent abdominal pain with nausea and sometimes vomiting. The symptoms may be present for months or years and the single attacks may be very severe. However, there is no strangulation or obstruction. No tumor masses can be felt, and there is no bleeding from the bowel.

FRANZ J. LUST

GASTROJEJUNOCOLIC AND JEJUNOCOLIC FISTULA. B. Isaacs. Glasgow M. J. 30:82-88, (March), 1949.

This is a report on two cases of gastrojejunocolic fistula following gastroenterostomy and one case of tuberculous jejunocolic fistula. The former condition may follow the perforation of a stomal ulcer into the colon. Ileosigmoidostomy gave remarkable relief to the first two cases.

Rapid emptying of the stomach and hyperperistalsis of the jejunum is the main factor in the production of resultant diarrhea. Due to this physiologic change a deficiency state develops.

A. X. ROSSIEN

MALIGNANT TUMORS OF THE SMALL INTESTINE. Irwin E. Siris. Am. J. Surg. 77:573, (May), 1949.

Four cases of malignant tumors of the small intestine have been presented. The underlying morbid process was different in each case. They illustrate the difficulty in early recognition and the reasons for the grave prognosis. 1) The early symptoms are vague intermittent abdominal distress and distention bearing no relation to meals; followed by progressive weakness and severe anemia. 2) Radiographically early, the disease is variously diagnosed and a correct interpretation is rarely made before the onset of partial obstruction. 3) With the onset of obstruction the disease is generally too far advanced to achieve a successful outcome. The disease frequently extends to the mesentery which is foreshortened. The lymphatic spread precludes radical extirpation of the inaccessible lymphatics. 4) Metastasis and early recurrence within a few weeks to two years is to be expected if thorough extirpation of involved lymphatics cannot be effected. The disease is resistant to radio therapy.

Extirpation of malignant tumors of the small intestine, even in advanced stages, is attended with a relatively low immediate operative mortality, therefore, earlier recognition of the disease may enhance the ultimate result. This may possibly be accomplished by 1) close evaluation of the history, 2) improvement in radiographic studies in order to ferret out the early intra- or extraluminary encroachments and 3) comprehensive repeated laboratory investigations, particularly for occult blood in the stools.

FRANZ J. LUST

LIVER AND BILIARY TRACT

REVIEW OF CERTAIN ASPECTS OF PATHOLOGIC PHYSIOLOGY OF CIRRHOSIS OF THE LIVER. W. A. Ricker. Northwest Med. 48:319-320, (May), 1949.

Fatty degeneration in the lobules of the periportal hepatic cells is the earliest distinguishable change of portal cirrhosis. This change is accompanied by some fibrosis between these liver cells, coming from the portal stroma. This has been demonstrated in the rat by administration of thorium dioxide which fixes the tissue macrophages. Lightly exposed x-ray films to these areas visualize progression or regression of the fibrous tissue. The fatty degeneration includes the hepatic cells; necrosis followed by regeneration develops in some instances. Advanced cases of cirrhosis may also include cellular infiltration, proliferation of small bile ducts and enlargement followed by shrinkage of the liver.

The initial damage to the hepatic cells produces the fatty change. Physiologically this change is due to dietary deficiency of those substances having lipotropic action (phosphorylation of neutral fats to phospholipidis). Hence cirrhosis in the human can be brought about by limiting the dietary intake of the lipotropic amino acids below critical levels. Choline plus cystine has the same lipotropic effect as methionine. Liver damage can be produced in rats within one hundred fifty days to the point of necrosis and/or cirrhosis by depriving the animal of methionine. Well established cirrhosis with scarring has been shown to be reversible in at least one instance. In the human, alcoholic and also dietary cirrhosis can be restored to normal with adequate intake of lipotropic substances.

About 50 per cent of patients have shown improvement on prescribed high protein and Vitamin B therapy. Persons with small livers do not respond when on a combination of choline and cystine therapy; those with large livers responded well, probably due to the large amount of fat in the liver. These are clinical findings.

On needle biopsy, only five of seven cirrhotic liver patients, repeatedly demonstrated progression of the fibrosis in spite of the clinical and functional improvement under lipotropic therapy given by Franklin and his co-workers in their series of fifteen patients.

Further investigation is necessary before there is definite establishment that an absorption of scar tissue in human cirrhotics occurs. A. X. Rossien

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SPLEEN

NONPARASITIC CYSTS OF THE SPLEEN. Irving H. Parnes. J. Mt. Sinai Hosp. 16:245, (Nov.-Dec.), 1949.

Two cases of cysts of the spleen are reported: one epidermoid in character, the other a false cyst of traumatic origin. Both were in young women. One patient had completed a normal pregnancy 18 months before onset of symptoms, which were ushered in by acute abdominal pain most probably due to a partial rupture of the spleen. The other patient had had infectious mononucleosis 7 months before onset of the recent symptoms, which consisted of dull and sharp pains in the left upper abdomen. Splenectomy was performed in both cases. The roentgenograms and the photos of the specimens are reproduced. Franz J. Lust

BOOK REVIEWS

THE 1950 YEAR BOOK OF MEDICINE. Paul B. Beeson, M.D.; J. Burns Amberson, M.D.; William B. Castle, M.D.; Tinsley R. Harrison, M.D.; George B. Eusterman, M.D. 819 pages. Illustrated. The Year Book Publishers, Inc., Chicago, Ill., 1950. Price $5.00.

The appearance of this volume in its new format is quite appealing. It is well written, illustrated. and the abstracts with their references convey to the reader invaluable saving of time and money. The busy practitioner will find what he is looking for, the latest in diagnosis, medication and therapy.

There are five subdivisions: Infections; The Chest; The Blood and Blood-forming Organs; The Heart and Blood Vessels and the Kidneys; and the Digestive Tract.

Each section is under the direct supervision of the chief editor who is an authority in his special field.

Dr. Beeson discusses infectious diseases in the decade 1940-50, thus bringing the reader up to date on this important phase of medicine. He mentions the various antibiotics, including terramycin (p. 36). On page 30 there is an interesting report on oral penicillin and its effect on preventing recurrences of rheumatic fever. The results after three years of observation should commend its more frequent use in children.

Page 34 should be read carefully because it discloses the fallacy advocated by pharmaceutical concerns or their detail men that aluminum hydroxide, given with aureomycin, alleviates the accompanying nausea and vomiting, at the same time it reduces aureomycin plasma concentration to one-fourth or to one-eighth. The reviewer found that frequent and small feedings, or milk, when not contraindicated is preferable to the aluminum hydroxide. This is also the opinion of the editor of this section.

On page 39 the reader will find a useful suggestion for aspirating tracheobronchial secretions which may cause asphyxial hazards in medical diseases or emergencies.

Penicillin treatment of diphtheria carriers is described on page 49.

Enteric infections from the American cockroach are reported by several investigators from the University of Texas. According to these investigators efforts should be made to eradicate these carriers.

Tuberculous peritonitis was treated by streptomycin in daily doses of 1-3 grams, intramuscularly in two to six divided doses. The striking and dramatic response to streptomycin was subsidence of fever. Abdominal tenderness, pain, etc., in most patients were relieved after one to two weeks.

On page 85, etc., the reader will find discussion of antihistamine drug therapy in the common cold. and the investigations carried out by the Council on Pharmacy and Chemistry of the American Medical Association.

Acute Herpetic Gingivostomatitis in adults is described on page 89. This is an interesting and timely essay. Many patients present these symptoms and the physician is puzzled as to the proper therapy.

There is an interesting article on complications following vaccine injections (p. 139), and numerous other abstracts which the reader will find useful in his daily practice.

On page 157 there is a special article on "Progress against diseases of the Chest" reviewing the past 10 years in this field of clinical medicine. In this chapter, as in the previous one, there are innumerable well written abstracts and hints on diagnosis and therapy.

In order that this review not be too lengthy the reviewer recommends the reader to peruse the other chapters with equal fervor.

Page 657 edited by Dr. Eusterman, a well-known clinician associated for many years with the Mayo Clinic, Rochester, Minnesota, introduces the subject of Gastroenterology with the statement that "undoubtedly greater medical progress has been made in the past half-century than in the previous 200 years". The reviewer agrees fully with this statement. Great progress had been made in the diagnosis and therapy of gastrointestinal diseases since Roentgen discovered his epoch-making "Roentgen-rays", which are utilized daily in the diagnosis of gastric pathology. Gastric analysis, gastroscopy. gastrophotography, peritoneoscopy and cytology of the gastric, biliary and pancreatic secretions in addition to attempts to evaluate the Papanicolaou and the Gladstone sponge method as a differential diagnosis aid in suspected carcinoma of the esophagus, stomach, rectum and colon.

In this chapter are discussed the latest findings, theories, and therapy related to diseases of the esophagus; the stomach and duodenum; the liver; pancreas and gallbladder and the intestinal tract.

On page 715 there is a clear and comprehensive description of the termination of the common bile duct. It is based on 50 dissections. On the opposite page half tone and line drawings illustrate the article.

Electrophoretic studies in portal cirrhosis are interesting as are more recent diagnostic aids in this condition.

Alleged adiposity of gallstone patients are discussed by Schidt of Denmark, and Alvarez describes hysterical type of nongaseous abdominal bloating which he considers to be caused by contraction of abdominal muscles, and often to assuming a lordotic posture which forces the abdomen forward.

Diagnosis of celiac disease and its treatment, according to Sidney V. Haas and Merrill P. Haas, is due to an altered intestinal function. They recommend a specific carbohydrate diet which incidentally is comprised of bananas and protein milk. The physician is referred to this interesting article which appeared in toto in Post Graduate Medicine, April 1950.

An excellent index and authors' roster complete the book.

THE 1950 YEAR BOOK OF RADIOLOGY. Edited by Jenner Hodges, M.D., Professor and Chairman, Department of Roentgenology, University of Michigan: John Floyd Hold, Associate, Department of Roentgenology, University of Michigan, Radiation Therapy: Isadore Lampe, M.D., Associate Professor, Department of Roentgenology, University of Michigan; and Robert S. Mac Intyre, M.D., Associate Professor, Department of Roentgenology, Universitv of Michigan. 480 pages. The Year Book Publishers, Inc., Chicago, Ill., 1950. Price $6.75.

As in previous years this volume on radiology and radiotherapy is up to expectations. It is beautifully printed, illustrated, and indexed. There is also an elaborate authors' index.

Part I deals with diagnosis and general technical developments. The abstracts and illustrations gleaned from the literature dealing with roentgenology brings to the reader all that is new and valuable in diagnosis.

Part II, dealing with radiation therapy, acquaints the nonspecialist with the technic, methods, application and results obtained by the use of radiotherapv, radium, mustard gas, iodine, etc.

The specialist in this field will find abstracts of the literature with adequate explanation as to technic and results.

The gastroenterologist will find a description (page 438), on "Rotation Treatment of Cancer of the Esophagus", by Carl Krebs et al, Denmark. This is an ingenious method which has been used on 137 men and 80 women with cancers of the esophagus and the cardiac end of the stomach. The results after treatment were quite obvious. It is to be hoped that this method of treatment will find favor in the United States.

This volume, as well as the others published by the Year Book Publishers has been of great help to the reviewer for more than four decades.

LES ICTERES. I. Pavel, Maître de Conférences à la Faculté de Médicine de Bucarest, Médecin de l'Hôpital Cantacuzino. 3rd Edition. 148 pages. Masson & Cie., Paris, 1949. Price 600 Francs.

This is an interesting monograph on "Ictères". The gastroenterologists, and clinicians, and surgeons who read French, will be interested in reading the contents of this monograph. There are a number of instructive illustrations, the reproductions of x-ray films are clear and understandable. As is to be expected, nearly all the references are from the French medical literature. Very little consideration—practically *none at all*—is given to the recent advances and studies reported in the extensive American, and English and Latin-American medical literature. Very few readers will buy this monograph—unless they are interested in reading a French contribution to the study of "Jaundice"!

HEMORRHAGIC DISORDERS. A GUIDE TO DIAGNOSIS AND TREATMENT. Paul M. Aggeler, M.D., Assistant Clinical Professor of Medicine and S. P. Lucia, M.D., Professor of Medicine, University of California Medical School. Lettered and illustrated by Phyluria Gibbs, Helene Cleare and Jean Thompson. 112 pages. The University of Chicago Press, Chicago, Ill., 1948. Price $10.00.

In preparing this rather unusual monograph the authors have had the cooperation of a number of their colleagues. The pages of the text were reproduced from an exhibit designed for use in teaching at the University of California Medical School. The authors give a concise summary of the physiology of hemostasis, the technics of hemostatic tests, the clinical features of disorders characterized by abnormal bleeding, and the agents and procedures which are available for treatment. The authors briefly, but instructivelv describe data concerning bleeding time, capillary fragility, platelet count prothrombin concentration, clot retraction measurement, and coagulation time. Among the conditions very briefly discussed are hemophilia, idiopathic hypoprothrombinemia, hemorrhagic disease of the newborn, hemorrhagic diathesis due to circulating anticoagulant, constitutional fibrinogenopenia: parahemophilia referring to Owren's case of a 29-year-old woman: hyperheparinemia; primary thrombocytopenic purpura including hypersplenism, secondary thrombocytopenic purpura, and hereditarv hemorrhagic telangectasia is presented with several illustrations, with eight references to the literature in the bibliography. The authors have omitted the earlier important references to Goldstein's articles 1921, 1930; Osler, 1907, 1911; Hanes. 1909; Legg; F. Parkes-Weber; Rendu, 1896; and Karl Ullman 1896, 1900, 1931. "Diseases of the Liver" and the hemorrhagic manifestations are covered in five pages. Gastrointestinal Diseases and hemorrhagic complications. are covered in four pages. Hypertension and Renal Disease—the hemorrhagic complications, are discussed in three pages, with illustra-

tions. The therapeutic agents and procedures appear in Section IV. Here one finds very brief comments on whole blood transfusion, anti-hemophilic globulin; thrombin; thromboplastin preparations; plasma transfusion; snake venoms; calcium salts, parahormone and Vitamin D, Congo red; dicarboxylic acids; nicotinic acid; protamine sulfate and toluidine blue; amino acids; pectin; Vitamin C; Vitamin K; hesperidin C; rutin; fibrin foam, gelfoam, oxycel; ultraviolet irradiation; roentgen irradiation of the spleen; splenectomy; Banti's syndrome, tuberculosis of spleen, reticuloendotheliosis of the spleen, Gaucher's disease; and selected cases of primary thrombocytopenic purpura. There is some discussion of the Use of the Anticoagulants, heparin and dicumarol (pages 92-96). A helpful bibliography of thirteen pages, concludes the text.

This book follows closely the pattern set by William Dameshek, of Boston, leading American hematologist, in his monographs "The Spleen and Hypersplenism", (1947), etc. This monograph is recommended to all medical students and general practitioners of medicine interested in hemorrhagic disorders. It should be helpful to all laboratory technicians.

MEMORIES OF EIGHTY YEARS. James B. Herrick, M.D. 270 pages. The University of Chicago Press, Chicago, Ill., 1949. Price $5.00.

Dr. Herrick, the experienced clinician, and well-known author of A Short History of Cardiology, and many scientific papers, including Coronary Thrombosis, has written a most interesting autobiography in fifteen chapters. There is evidence in this refreshingly interesting story of the cultured gentleman, the keen observer, and the humanitarian.

The reading of this book is a source of inspiration, and pleasurable relaxation.

Dr. Herrick was born August 11, 1861, and in his 89th year, he clearly recalls the funeral procession of Abraham Lincoln in Chicago, in April 1865, when Herrick was only nearing 4 years of age. He was graduated from Oak Park High School in 1877, and attended Rock River Seminary, and University of Michigan at Ann Arbor, where he became a member of Psi Upsilon. He was graduated in 1882. Herrick was a high school teacher 1883-1886. Dr. Herrick received his medical degree at Rush on February 21, 1888, sixty-three years ago. He spent some time at Cook County Hospital, and then became assistant to Dr. C. W. Earle. He began to receive office patients in the basement of his home in cramped quarters on November 1, 1890, just sixty years ago!

In Chapter VII, the author gives an interesting account of his studies abroad—in Prague, Erlangen, Heidelberg, Munich, Berlin, Würzburg, and Vienna, and here, includes excellent photographs of the Herrick family, Herrick as a house surgeon, Cook County Hospital Staff; Hans Chiari; Emil Fischer; Edmund Neusser; Nicholas Senn; J. B. Murphy; Frank Billings; Christian Fenger; Bertram W. Sippy; Dean Lewis, and Fred M. Smith. The frontispiece gives an excellent likeness of the author at seventy-five. He reviews his contributions to medical literature in Chapter XI including "Reports of Cases with Autopsies"; "Clinical Features of Sudden Obstruction of the Coronary Arteries", based on a case of coronary thrombosis in which he made an antemortem diagnosis; "Coronary Thrombosis"; "An Intimate Account of My Early Experiences with Coronary Thrombosis". The closing Chapter XV, begins with the author's birthday, August 11, 1948 at the age of eighty-seven years, and describes his "Old Age". "I don't like old age and am not afraid to say so", quoting Samuel Johnson and agreeing with him, when he says, referring to old age as a period "in which there is much to be evidenced and little to be enjoyed". The author quotes Chesterfield, who in his old age said: "Tyrawley and I have been dead these two years, but we do not choose to have it known". Herrick does not like the loss of memory for recent events in old age—although, for years he had an excellent memory, and he comments about his early retentiveness of memory. He recalls clearly many events of years gone by. Recalling Tennyson's "Ulysses", who could say to his fellow-mariners: "You and I are old; old age has yet his honor and his toil; death closes all; but something ere the end, some work of noble note, may yet be done". The sundial in the garden counts only the sunny hours—let us do likewise; and, still hear the voices of the singing birds, and the laughter of happy children in our aging years!

Thus speaks a lovable and much beloved American doctor of the old school—let us all follow in his wise teachings and, sitting at his feet, be inspired to higher ambitions and a brighter outlook for the future of medicine! This excellent autobiography should become popular reading for physician and layman, medical student, interne, and nurse.

POSTGRADUATE GASTROENTEROLOGY. Edited by Henry L. Bockus, M.D., Professor of Gastroenterology, University of Pennsylvania Graduate School of Medicine. 670 pages. W. B. Saunders Co., Philadelphia, Pa., 1950. Price $10.00.

Numerous members of the Faculty and contributors have made this volume an excellent treatise on Postgraduate Gastroenterology which was given under the sponsorship of the American College of Physicians in Philadelphia, December 1948.

In glancing over the contents one wonders how it was possible in 6 days to cover the ground of the entire gastrointestinal tract including panel discussions and presentation of cases at gastroenterological conferences.

The physician and student is recommended to read carefully the interesting discussion on pyrosis (heartburn). This paper was published in the Journal of the A.M.A., and those who missed it will find this repetition a valuable exposition of the mechanism and clinical manifestation of this oft appearing symptom.

It would take reams of paper to describe the other presentations. All in all it is a valuable addition to the medical literature and is highly recommended as a must for the physician's reference library.

NORMAL VALUES IN CLINICAL MEDICINE. F. William Sunderman, M.D., Ph.D., Professor of Experimental Medicine and Clinical Pathology, University of Texas Postgraduate School of Medicine; and Frederick Boerner, V.M.D., Late Associate Professor of Clinical Bacteriology, Graduate School of Medicine, University of Pennsylvania. 845 pages, 237 figures, and 413 tables. W. B. Saunders Co., Philadelphia, Pa., 1949. Price $14.00.

There are forty-four collaborators, most of them associated with the University of Pennsylvania. Most of these collaborators have contributed numerous scientific papers and have enriched the literature with their observations.

The book is divided into nineteen sections, seventy-four chapters, an appendix, and index. In addition, numerous graphs, illustrations, tables and an adequate bibliography add to its value. It is hoped that physicians will find valuable information regarding "normal values" which up to the present time was an impossibility owing to individual "norms" established by clinicians and investigators.

For the general practitioner this volume will be a handy reference, for the specialist in the various specialties, eye, ear, nose, internal medicine, normal values will help to establish diagnosis in borderline cases.

The authors, publishers, and collaborators deserve the thanks of the medical profession for publishing such a beautiful and comprehensive volume in which the text, illustrations, and last but not least, the format is everything which is to be desired.

THE CYTOLOGIC DIAGNOSIS OF CANCER. The Staff of the Vincent Memorial Laboratory of the Vincent Memorial Hospital. A Gynecologic Service Affiliated with the Massachusetts General Hospital, Boston, Massachusetts. The Department of Gynecology, Harvard Medical School. Published under the Sponsorship of The American Cancer Society. 229 pages with 153 figures. W. B. Saunders Co., Philadelphia, Pa., 1950. Price $6.50.

The dedication of this book to Dr. Papanicolaou by its authors is a tribute to one who pioneered in the cytologic diagnosis of cancer. Originally, Dr. Papanicolaou studied vaginal smears and in 1941 published his first clinical paper in the early recognition of cancer of the cervix and endometrium.

This volume is the outcome of tedious and arduous work by the Vincent Memorial Laboratory Staff affiliated with the Massachusetts General Hospital. Not only is the uterus and vagina included in these studies, but the organs of respiration, digestion, and urinary tract have also been carefully observed and as the reader will find, beautiful black and white and colored plates enhance the explanatory text.

It is highly recommended as a reference book for both the physician and research worker.

CLINICAL PATHOLOGY: Application and Interpretation. Benjamin B. Wells, M.D., Ph.D., Professor of Medicine, University of Arkansas School of Medicine, Little Rock, Arkansas. 397 pages with 32 figures. W. B. Saunders Co., Philadelphia, Pa., 1950. Price $6.00.

A well-written and illustrated text dealing with a difficult subject should be a welcome edition to clinicians and pathologists.

Nine chapters, appendix and extensive cross-index are features which add greatly to the value of the book, the type is clear and the illustrations and tables are explanatory.

Chapter 1 deals with infectious diseases and their recognition by means of laboratory procedures. One hundred and eleven pages are devoted to this feature.

The gastrointestinal tract studies in Chapter 2 cover the subject adequately. Oral and pharyngeal signs of disease, ulcer, cancer, etc., of the stomach and duodenum, the biliary system, pancreas, small and large intestinal diseases including sprue are discussed in detail.

Chapter 3 deals with respiratory diseases; Chapter 4, diseases of the kidney and urinary tract; diseases of the blood is found in Chapter 5; cardiovascular, metabolic and endocrine disorders, complete Chapters 6 and 7.

Chapters 8 and 9 deal with laboratory studies in surgery and in obstetrics.

In the appendix, on page 377, the reader will find valuable information diagnosing normal and abnormal states of the blood, sputum, cerebrospinal fluid, etc.

DISEASES OF THE HEART. Charles K. Friedberg, M.D., Associate Physician, Mt. Sinai Hospital, New York; Lecturer in Medicine, Columbia University. 1081 pages with 79 figures. W. B. Saunders Company, Philadelphia, Pa., 1949. Price $11.50.

This excellent book does "provide a comprehensive and integrated exposition of the diseases of the heart". Pathologic physiology and the pathogenesis of the symptoms and signs of cardiac disorders, are most instructively covered by the experienced author. This textbook on diseases of the heart is one of the best books now available for all practitioners of medicine, specialists, and medical students who are interested in knowing more and more about this increasingly important branch of medicine. This book may well be kept for ready use on every physician's desk, whatever his medical interests are, and whatever his particular specialty may be. There are few books now appearing, that can even come close to this one by Doctor Friedberg!

OPERATIONS OF GENERAL SURGERY. Thomas G. Orr, M.D., Professor of Surgery, University of Kansas School of Medicine, Kansas City, Kans. Second Edition. 890 pages with 1700 step-by-step illustrations on 721 figures. W. B. Saunders Company, Philadelphia, Pa., 1949. Price $13.50.

This second edition of Dr. Orr's volume on Surgery surpasses the original publication. The publishers spared no expense in making this volume a real teaching text. The third year medical student who is being initiated into the mysteries of clinical minor surgery will not have any excuse that he is unable to grasp the principles of minor surgery. Later as he becomes a senior, clinical assistant, interne and resident, he will find the step-by-step illustrations and explanations both in minor surgery, major surgery and the specialties, clear and to the point.

It is highly recommended as a practical guide to the student as well as the surgeon. The latter will find that the illustrations and texts will aid him in his chosen work.

CLINICAL BIOCHEMISTRY. Abraham Cantarow, M.D., Professor of Biochemistry, Jefferson Medical College; and Max Trumper, Ph.D., Commander, H(S), USNR, Lecturer in Clinical Biochemistry and Basic Science Coordinator, Naval Medical School, National Naval Medical Center, Bethesda, Md. Fourth Edition. 642 pages with 38 figures. W. B. Saunders Company, Philadelphia, Pa., 1949. Price $8.00.

The present edition has been extensively revised and the text brought up to date to include thymol turbidity and flocculation and adrenocortical function studies.

A complete index and table of contents add to the value of the book.

The student and physician will find it a valuable asset in their daily contact with patients.

AN ATLAS OF THE BLOOD AND BONE MARROW. R. Philip Custer, M.D., Director Laboratories of the Presbyterian Hospital in Philadelphia; Assistant Professor of Pathology, The University of Pennsylvania School of Medicine; Consultant to the Armed Forces Institute of Pathology. 321 pages with 285 figures, 42 in color. W. B. Saunders Company, Philadelphia, Pa., 1949. Price $15.00.

This Atlas of Blood and Bone Marrow is a most comprehensive and complete volume on this important subject. The illustrations and text are clear and differ from the usual run-of-the-mill texts. Almost all of the photographs are taken from original sources rather than from artists' drawings.

There is a comprehensive and extensive index which enhances the value of the volume.

The author and the publisher are to be congratulated on bringing out this important book.

STUDIES OF TUMOUR FORMATION. G. W. de P. Nicholson, M.D., Professor of Pathology, Guy's Hospital, London. Foreword by Rupert A. Willis, Introduction by S. De Nevasquez. 637 pages. The C. V. Mosby Co., St. Louis, Mo. Price $15.00.

The source of most of Professor Nicholson's material was the surgical wards of Guy's Hospital, where he was Clinical Microscopist. Professor Nicholson was an experienced pathologist, and unsurpassed in tissue diagnosis. Nicholson's twenty tumour studies appeared in the Guy's Hospital Reports 1922-1938, and are classics indeed—these tumour studies are now reprinted in the original form in this excellent volume so that all who wish, may read and study the many carefully presented observations and comments, which abound in rich "cogent reminders that pathology is a branch of biology" (Willis). Expertly discussed in this work in a most informative and instructive manner, are: the morphology of tumours, tissue malformations, cutaneous moles, the hyperplasias, embryonic tumours of the kidney, heterotopic tumours, osteo- and chondro-sarcomata, endometrial tumours, teratomata; causation, reaction and environment; method in oncology; somatic development and tumour formation; and, ovarian goitre (Chapter 19) covers 47 pages; "Induction and Determination" (Chapter 20) completes the book, with his closing "Excursus". This is an unusual book—written by a medical philosopher and an honest and fearless expert pathologist.

THE PRACTICE OF MEDICINE. Jonathan Campbell Meakins, C.B.E., M.D., LL.D., D.Sc. Formerly Professor of Medicine and Director of the Department of Medicine, McGill University; formerly Physician-in-chief, Royal Victoria Hospital, Montreal; formerly Professor of Therapeutics and Clinical Medicine, University of Edinburgh. Fifth Edition. 1558 pages, 518 illustrations, 50 in color. The C. V. Mosby Co., St. Louis, Mo., 1950. Price $13.50.

The Fifth Edition of this textbook on the practice of medicine has been extensively revised and brought up to date. Many changes and great advances in medicine have been made since 1936, when this book first appeared.

This edition of 1950 is the best of the series. The author, an experienced clinician, and Editor of the American Heart Journal, has somehow, found time to keep up to date in nearly all phases of medicine.

Chapters X, XI, XII, and XIII (pages 586-887) consisting of 300 pages, cover in an informative manner, quite adequately for the general practitioner of medicine and the medical student and resident physician—"Diseases of the Gastrointestinal Tract", "Diseases of the Liver and Bile Passages", "Diseases of Nutrition", and "Diseases of Metabolism". These chapters are recommended for reading by general practitioners and gastroenterologists.

Other chapters of particular interest are those on "Diseases of the Ductless Glands" by Dr. E. H. Mason, (Chapter XIV, pages 888-973), "Diseases of the Nervous System" by the late Dr. J. N. Petersen (Chapter XV, pages 974-1160) and "Psychosomatic Medicine" by Dr. F. R. Hanson (Chapter XVI, pages 1161-1204). "Infectious Diseases" are well discussed and adequately presented in Chapter XIX (pages 1299-1418). Coccidioidomycosis, histoplasmosis, toxoplasmosis, Q-fever, Reiter's Disease, are lightly touched upon. Stevens-Johnson Syndrome, Hereditary Hemorrhagic Telangiectasis (Rendu-Osler-Weber's Disease) or Heredofamilial Angiomatosis, and a number of other clinical entities of interest, do not appear listed in the Index (pages 1503-1558). The section on "Diseases of the Circulatory System" (Chapter VII, pages 323-489) includes (118 lines of reading matter, pages 121-128), all-too-brief discussion on "Congenital Heart Disease", in view of the great advances made during the past decade in the surgical management of congenital cardiovascular conditions! This fifth edition of Meakins' "Practice of Medicine" is an improvement of this work, and is recommended for medical students and general practitioners of medicine, internes, and resident physicians.

GASTROENTEROLOGIA. Published by the Asociacion Nacional de Gastroenterologica, Bogóta, Colombia. No. 1. 1950.

This volume consists of papers presented at the meetings of this gastroenterological society. They are well written, the illustrations are clear and the text includes the esophagus, stomach, intestines, liver and the rectum.

The Editorial Board of THE REVIEW OF GASTROENTEROLOGY compliments the editors of this new Journal on Gastroenterology.

THE PHYSIOLOGIC BASIS OF OPERATIONS FOR DUODENAL, GASTRIC AND GASTROJEJUNAL ULCER. Henry W. Mayo, Jr., B.A., M.D., M.S., Associate in Surgery, Medical College of the State of South Carolina. With Foreword by Owen H. Wangensteen, M.D., Ph.D., Professor of Surgery, University of Minnesota Medical School. 86 pages. The C. V. Mosby Co., St. Louis, Mo., 1949. Price $3.50.

This work represents a portion of a thesis presented to the Graduate Faculty of the University of Virginia in Candidacy for the degree of Master of Science. It is unfortunate that this monograph is only "a portion" of the thesis—why not the whole complete thesis? There is an exhaustive alphabetically arranged bibliography of 541 references—from I. Abell to R. Zollinger, covering many of the important contributions to the literature on the subject of "ulcer", that have appeared during the past twenty-five or thirty years. The reviewer, who has carefully followed the "gastroenterologic" literature for the past forty years of his interest in the subject, fails to note many names.

This is a scholarly monograph which must have taken much of the author's time and energy. This little volume, on a very timely topic, is a worthwhile addition to one's library, and the fine bibliography may frequently be of considerable help to writers on the subject of "ulcer".

VARICOSE VEINS. R. Rowden Foote. 226 pages, 181 illustrations and 2 color plates and a color-frontispiece. The C. V. Mosby Co., St. Louis, Mo., 1949. Price $8.00.

Chapter I deals interestingly with "Some Historical Landmarks of Varicose Veins", in twenty-one pages, that are well worth reading by all students and physicians, and by medical historians. Anatomy, physiology, and pathology are considered in Chapter II (15 pages). Among the topics ably and expertly discussed by the author are: "Incidence and Etiology of Varices", "Investigation of the Patient—"; and "Treatment" (Chapter V) is well covered in fifty-five pages.

The author considers concisely, but adequately, "Varicose Ulceration" (Chapter VI); "Thrombophlebitis and Pulmonary Embolism" (21 pages); Anticoagulant Therapy-Heparin and Dicumarol are briefly discussed. The newest and most recent anticoagulant preparations will no doubt be included in the next revision of this fine monograph. "Supportive and Compressive Treatment"; "The Varicose Vein Clinic and some useful Prescriptions"—The Organization of the Clinic; and "Recent Work in Varicose Veins"—form the last three chapters of this very readable monograph—that is recommended to all practicing physicians, and surgeons—obstetricians, gynecologists, and industrial ("Occupational Medicine") physicians,—as well as Army and Navy physicians.

TEXTBOOK OF ANATOMY AND PHYSIOLOGY. Carl C. Francis, A.B., M.D., Assistant Professor of Anatomy, Western Reserve University, Cleveland, Ohio; and C. Clinton Knowlton, Ph.D., Assistant Professor of Physical Medicine, Emory University Medical School. Second Edition, 624 pages, 365 text illustrations and 31 color plates. The C. V. Mosby Co., St. Louis, Mo., 1950. Price $6.25.

This edition is a great improvement over the first edition. Additional illustrations and a number of replacements, and new color plates add to the instructive value of this excellent compact book on anatomy and physiology.

This work should be of considerable aid to all premedical and medical, and dental students, to all graduate and student nurses, and, will be quite adequately informative for all college students who are interested in this subject—as well as students of art, drawing and painting, and students of physical education—and instructors at college, in gymnasia, and in the armed forces, will profit by reading this book!

LEHRBUCH DER ROENTGENOLOGISCHEN DIFFERENTIAL DIAGNOSTIC-ERKRANKUNGEN DER BAUCHORGANE. Werner Leschendorf, M.D. Second Edition. 608 pages, 1081 illustrations. George Thieme Verlag, Stuttgart, Germany, 1950. Price $17.30.

Judging by the excellent quality of the printing and illustration this second edition of this well-gathered-up volume should be translated into English for those physicians who are unfamiliar with the German language.

In glancing over the contents the reviewer is impressed with the wealth of useful information which may help in the diagnosis of confusing or simulating conditions. In Chapter one, page 2, the author discusses and illustrates the coincidence of more than one cause for the patient's complaint, for example, the first illustration shows the presence of a gastric ulcer and kidney stone; the second and third illustrations show in the same patient a multiplicity of pathology-hiatus hernia, pyloric ulcer and a diverticulum in the duodenal-jejunal area.

Page after page the reader will find abundant descriptive and illustrative material and discussions of differential diagnosis not only on the anatomic but also on the etiologic basis.

Chapter two deals with the differential diagnosis (roentgenologically) of affections of the stomach. The text, illustrations, roentgen as well as the line drawings are explanatory. On page 126, the author calls attention to the possible errors in diagnosing hourglass or spastic condition of the stomach. He mentions various extragastric causes which may cause or simulate pseudo-hourglass, spastic states or irregularities including those found on the greater curvature.

At the bottom of page 128 he mentions Dinklin's method of establishing the site of a bleeding area (ulcer niche) in the stomach. The procedure is to give a mixture Hydrogen peroxide (3 per cent) 75., milk or water 200., Barium 100., White of egg 1, Gelatine 1:100, 10. The reviewer would hesitate to subject the bleeding patient to this procedure, although Schatzki advocates a thin mixture of barium to be given to a bleeding patient to facilitate localization of the ulcer.

Chapter three deals with differential diagnosis of the operated stomach. Chapter four describes the findings in duodenal conditions. Chapter five—differential diagnosis of cholecystopathies. Chapters six, seven, and eight deal with the other abdominal viscera, while chapter nine devotes more than fifty pages to artificial pneumoperitoneum in differential diagnosis of the liver, pancreas, spleen and tumors of the abdominal cavity.

In addition to the well written and illustrated text, there is a complete index at the end of the book and marginal notations on every page for rapid reference. This marginal notation would be of advantage in our texts also, but alas, the publishers feel that it is a waste of space and money.

Every physician interested who can read German will find this book a valuable addition to his library.

ELECTROCARDIOGRAPHY, FUNDAMENTALS AND CLINICAL APPLICATION. Louis Wolff, M.D., Visiting Physician, Consultant in Cardiology and Chief of the Electrocardiographic Laboratory, Beth Israel Hospital; Associate in Medicine, Harvard Medical School. 185 pages. Illustrated. W. B. Saunders Company, Philadelphia, Pa., 1950. Price $4.50.

This excellent, practical and very readable and understandable monograph on electrocardiography by an experienced teacher of the subject at Harvard Medical School is highly recommended for all

medical students, graduate students, internes and resident physicians, and, also, for clinicians, general practitioners of medicine, and cardiologists.

In this monograph, the author discusses briefly, but adequately and informatively, most of the basic principles of electrocardiography, "Unipolar and Bipolar Leads", "Intrinsic and Intrinsicoid Deflections", "Anatomy and Physiology of the Heart", "Primary and Secondary T-Wave Changes", "The Precordial Electrocardiogram", "The Einthoven Equilateral Hypothesis", right and left bundle branch block, left and right ventricular hypertrophy, and myocardial infarction (Chapter 17) is interestingly presented, in Part I of this little monograph.

In Part II the author presents in a clear and well-ordered manner the various abnormal electrocardiograms and furnishes the student with a fine discussion of "The Normal Electrocardiogram" (Chapter 19).

In the closing chapter (26) is presented "Short P-R Interval with Abnormal QRS Complexes. Wolff-Parkinson-White Syndrome". Lacking in this small monograph are one or two chapters on the electrocardiographic changes in the several important cardiovascular anomalies, and, in other disorders of the chest, including the electrocardiographic changes associated with certain types of chest deformities, tuberculosis (after operative procedures), hiatus hernia, etc.

The reviewer feels that this practical monograph for students and practitioners, and for those preparing for board examinations, may deservedly be placed on the shelf along with other monographs on the subject.

CARDIOVASCULAR DISEASE: Fundamentals, Differential Diagnosis, Prognosis and Treatment. Louis H. Sigler, M.D., F.A.C.P., Attending Cardiologist and Chief of Cardiac Clinic, Coney Island Hospital; Consulting Cardiologist, Rockaway Beach Hospital; Consulting Cardiologist, Menorah Home and Hospital for the Aged. 551 pages. Grune and Stratton, New York, 1949. Price $10.00.

The author, who is an experienced cardiologist, and also, primarily a clinician, who realizes that human cells do not always conform to rules and mathematical formulae, has written a very practical and most useful book.

All too often, have medical practitioners become the owners of electrocardiographs, fluoroscopes, and expensive x-ray machines, without the extensive and intensive training necessary for the development of that high degree of knowledge and expert judgment required for the proper use of these instruments, and the correct interpretation of the many "variables", and unusual findings, quite often misinterpreted by the inexperienced! Not infrequently do we hear of tragic errors of omission and commission, resulting from such misinterpretation of x-ray films, fluoroscopic studies, and electrocardiograms!

The book by Doctor Sigler should go far in preventing such errors. We have recently seen a good example of an erroneous reading of an electrocardiogram because of a technician's mistake in reversing the electrodes. The book consists of 31 chapters covering the general incidence of cardiovascular disease, etiology, essential features, physiologic principles of circulation, informative discussion of the normal sized heart and measurements and cardiac enlargement and dilatation of great vessels, normal and abnormal heart rate and rhythm, as well as normal and abnormal heart sounds, cardiovascular murmurs and normal and abnormal arterial and venous pulse.

The author satisfactorily and adequately discusses blood pressure, heart failure, angina pectoris, arterial hypertension and pulmonary vascular hypertension; coronary occlusion and hypocardial infarction, rheumatic heart disease, bacterial endocarditis and "chronic cardiovascular disease". "Congenital heart disease" is presented briefly in Chapter 28 (21 pages). Only a few of the more common forms of congenital heart disease are discussed. Doctor Sigler, in 1919, reported a case of coronary thrombosis in a man that he had diagnosed during life. The report was accompanied by electrocardiographic findings, and the diagnosis was proved at autopsy.

Psychosomatic Cardiovascular Abnormalities, Pregnancy and Cardiovascular Disease, and Surgery and Cardiovascular Disease are the titles of several chapters, in which these subjects are briefly, but instructively presented.

"Diseases of the Pericardium" are discussed in Chapters 25 and 26.

The author has published another monograph on electrocardiography, "The Electrocardiogram", a book of 415 pages. So that there are no electrocardiograms nor discussions of electrocardiographic abnormalities in this book. This work is recommended for the use of general practitioners, medical students, clinicians, and cardiologists. This volume should be readily available in all hospital-staff libraries for the use of the resident physicians and internes and members of the junior staff.

Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below relates gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro-intestinal	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifesta-tions	Palpitation Tachycardia Elevated B. P. Dry mouth—throat	Heartburn Nausea-vomiting Low B. P. Colonic spasm

Data here tabulated is from references 3,4,5,6,7, given below.

Diagnosis of functional disorder is supported by the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relief of symptoms by drug therapy (so making the patient more amenable to psychotherapy); 2) psychotherapeutic guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

Clinicians who have studied these disorders, including those of the menopause, report that good therapeutic results are produced by combined adrenergic (ergotamine) and cholerinergic blockade (Bellafoline) with central sedation (phenobarbital) [8,9,10]. A convenient preparation of this nature is available in the form of **Bellergal Tablets.** Functional disorders are long-term therapeutic problems; therefore, drug treatment by the following method is recommended: 5 or 6 tabs. per day for the 1st week; then gradually reduce to the smallest dose effective in maintaining the patient symptom free (average: 3 tabs. daily). Interrupt for 1 week out of every month to assess results.

1. Ebaugh, F.: Postgrad. Med. *4:* 208, 1948. 2. Wilbur, D.: J.A.M.A. *141:* 1199, 1949. 3. Williams, E. and Carmichael, C.: J. Nat'l. Med. Assoc. *42:* 32, 1950. 4. Goodman, L. and Gilman, A.: The Pharmacological Basis of Therapeutics, The Macmillan Co., 1941. 5. Katz, L. et al: Ann. Int. Med. *27:* 201, 1947. 6. Weiss, E. et al: Am. J. Psychiat. *107:* 264, 1950. 7. Alvarez, W.: Chicago Med. Soc. Bulletin, 581, 1950. 8. Rakoff, A.: A Course in Practical Therapeutics, Williams and Wilkins, 1948. 9. Karnosh, L. and Zucker, E.: A Handbook of Psychiatry, C. V. Mosby Co., 1945. 10. Harris, L.: Canad. M.A.J. *58:* 251, 1948.

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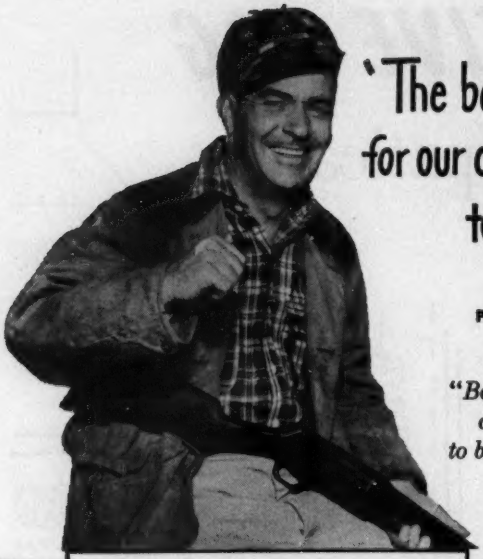

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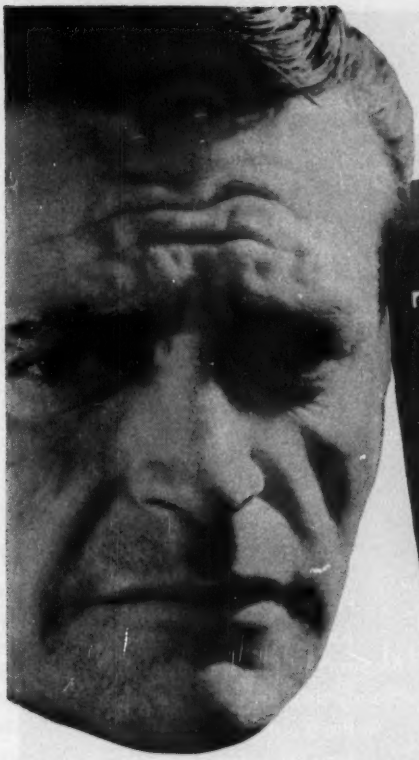

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*Offenkrants., W. F., Rev. Gastroenterol, 17:359-367 (May). 1950

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[1]Seley, S. A.: Medical Management of Pyloric Obstruction Resulting from Peptic Ulcer, *Am. J. Dig. Dis., 13*:238, 1946.

*T. M. Reg. U. S. Pat. Off.

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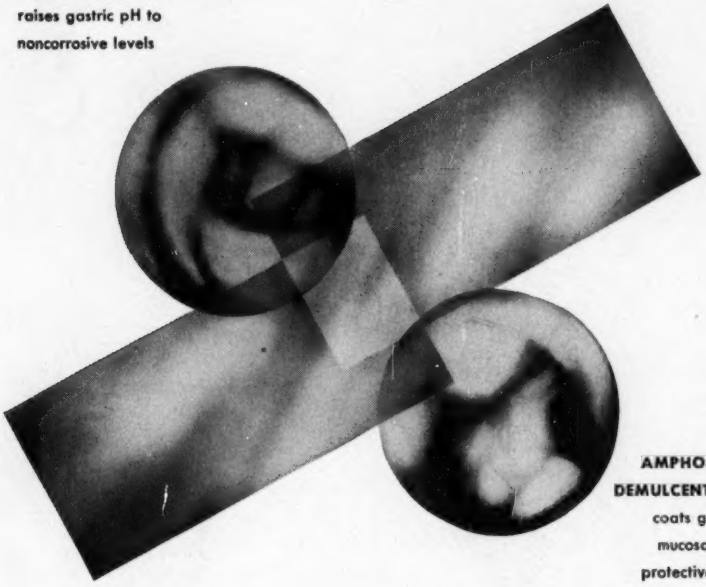

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